THE SURGICAL CLINICS OF NORTH AMERICA

FEBRUARY 1974

VASCULAR SURGERY

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SYMPOSIUM ON
VASCULAR SURGERY

John J. Bergan, M.D., and
James S. T. Yao, M.D., Ph.D., *Guest Editors*

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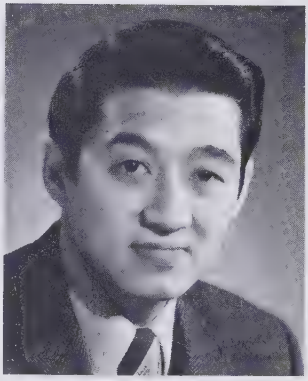
JOHN J. BERGAN



JAMES S. T. YAO

This volume touches on the future of vascular surgery. It is concerned with the training of the vascular surgeon, describes instrumentation and techniques for quantitation of blood flow, and details new methods for dealing with old vascular problems. De Weese's essay on training of the vascular surgeon is particularly timely. This is an era of great interest in certification of vascular surgeons.

Responsibility for presentation of this material has been spread among representative vascular surgeons in Chicago, the United States, England, and Europe. The chosen authors have proven particularly articulate.

Among the articles which deal with applications of biophysics in peripheral vascular diagnosis are descriptions of ultrasound by Strandness and by Yao, and isotope techniques by Lassen of Copenhagen and Quinn of Chicago. Connolly from Irvine has succeeded in setting forth the current status of impedance plethysmography in vascular diagnosis and has correlated this with clinical findings.

Innovative methods used to prevent amputation in severe ischemia of the foot are detailed. The large experience in femorotibial grafting accumulated by Bernhard of Milwaukee is as notable as the intriguing

studies of arterial endoscopy by Vollmar. Peter Martin's experience with profundaplasty is presented as a valuable adjunct in direct arterial surgery and Trippel summarizes very well the place of extra-anatomic reconstructions in patients with severe ischemia. The observations made by Taylor and by Downs on femoropopliteal and aortoiliac operations are particularly cogent.

Common problems of vascular reconstruction are solved in a fresh manner by several authorities in the last section of this volume. Of special interest is the presentation by Sauvage on future possibilities in prosthetic materials and the philosophical approach to thromboembolism stated by Professor Browse of St. Thomas' Hospital. The large experience in treating renovascular hypertension accumulated by Foster is summarized as is the current surgical approach used by Javid and his group in treatment of cerebral ischemia.

Of particular use to the general surgeon who has an interest in vascular problems are Fry's views on treatment of arteriovenous fistulae, Conn's thoughts on thoracic outlet syndromes, and Clinton Compere's methods of rehabilitation of the elderly amputee.

The Editors have enjoyed the extraordinary opportunity of bringing to the surgical reader this compilation of new developments in vascular surgery. The past several years have been an exciting time in surgical treatment of vascular disease.

JOHN J. BERGAN, M.D.
JAMES S. T. YAO, M.D., Ph.D.
Guest Editors

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Training and Certification of Vascular Surgeons

*James A. DeWeese, M.D.**

In 1946 operations for the treatment of vascular diseases were common enough that a small group of interested surgeons formed the Society for Vascular Surgery. One of the objectives of the Society was to define more clearly the role of surgery in vascular disease. At that time there were a limited number of appropriate operations. For example, the only operative procedures discussed at the first meeting of the Society in 1947 were femoral vein ligations, ligations and stripping of the greater saphenous vein, porta caval shunts, lumbar and thoracolumbar sympathectomies, and the correction of coarctation of the aorta. Since 1947 there has been an extremely rapid growth in the number and types of vascular operations. A list of the type of operations now performed includes:

- Reconstruction of arteries
  - Extracranial cerebral arteries
  - Visceral arteries
  - Aortoiliac arteries
  - Lower extremity arteries—usually femoropopliteal
- Resection and replacement of aneurysms
  - Abdominal aortic
  - Peripheral arteries
  - Visceral arteries
- Embolectomy and thrombectomy
- Treatment of arteriovenous fistulae
- Sympathectomy
- Amputation
- Thoracic outlet decompression
- Venous interruption
- Venous reconstruction
- Venous ligation and stripping
- Operation for lymphedema
- Portal venous decompression

These operations are now being performed by many different specialty groups with considerable local and regional variations. In some areas sympathectomies and carotid endarterectomies are performed by

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neurosurgeons, amputations for gangrene and thoracic outlet decompressions are performed by orthopedic surgeons, and operations for lymphedema are handled by plastic surgeons. In most communities general surgeons or thoracic and cardiovascular surgeons are performing the operations. It is the author's observation through discussions with former residents and other surgeons throughout the country, that an increasing percentage of major vascular operations are performed by surgeons who devote a significant amount or all of their time to the treatment of vascular diseases.

On the other hand, a considerable number of vascular operations are still being performed by the surgeon who does only an occasional case. This contention is supported by a survey carried out by the American Hospital Association indicating that during 1969 fewer than 10 elective abdominal aortic aneurysm resections were performed in 75 per cent of the hospitals reporting any that year.[9] In addition it is estimated that 37 per cent of all surgical operations in the United States are performed by surgeons not in training but without surgical board certification.[7] These observations raise the possibility that many vascular operations are performed by individuals without training in vascular surgery or even without complete training in general surgery or any surgical specialties, who are performing only an occasional vascular operation.

Suboptimal results from vascular operations secondary to lack of experience of the surgeon have been the subject of concern to Cannon and Wylie in their presidential addresses before the North American Chapter of the International Cardiovascular Society.[1, 14] Their concern is shared by many others. Hanlon in his presidential address before The Society for Vascular Surgery also challenged its members to strive for higher standards of excellence.[4] With this background the membership of The Society for Vascular Surgery and The North American Chapter of the International Cardiovascular Society passed a resolution at their annual meeting in 1972 recommending and endorsing a certification method for recognition of special competence in vascular surgery under the aegis of the American Board of Surgery. It is fitting that these two Societies provide leadership in these areas, since they both accept responsibility for the dissemination of knowledge regarding the treatment of vascular diseases. In addition, the objectives of The Society for Vascular Surgery include the promotion and encouragement of adequate teaching of vascular disease to students and house officers as well as the encouragement of hospitals to develop special training for young surgeons interested in the field.

It is of some interest that, at this time, the training and certification of vascular surgeons is also of international concern. Vollmar has suggested that departments of vascular surgery be developed in the hospitals of Western Germany.[12] Deterling in his presidential address before the meeting of the International Cardiovascular Society in Moscow in August of 1971, suggested the organization of a committee on international education to "develop standards in training and practice of vascular surgery."[3] With this background, let us then consider the status of vascular surgery training and certification in the United States.

TRAINING

Competent vascular surgeons have obtained their training in several different ways. There are still many pioneers in vascular surgery, particularly in the area of vascular reconstructions, who are actively practicing. These surgeons and their associates, in general, learned, practiced, and modified their surgical techniques in the laboratory. Once they mastered vascular anastomoses and found workable materials for vascular replacements, the operating room became the laboratory for the application of the technique for replacing, bypassing, or endarterectomizing these blood vessels. Mistakes were made but fortunately intelligent evaluation of errors, improvement of techniques, and increasing experience decreased their occurrence. There need not be and should not be continued use of trial and error by surgeons untrained in vascular surgery and performing only an occasional vascular operation to relearn the lessons already known by competent vascular surgeons.

When vascular surgery was previously defined and later discussed, we spoke of vascular surgery primarily in terms of vascular operations and particularly vascular reconstruction. This operation has been overemphasized to point out that the margin for error in vascular surgery is less than in most fields. Errors in technique often result in irreparable damage to organs or loss of an extremity and second chances are frequently not available. Skill and judgment, which may be equally as important, are rewarded by good results but the procedures, in general, are unforgiving of errors. Good judgment is not only important in the operating room, but is equally important in the preoperative period. A vascular surgeon requires a detailed knowledge of all vascular diseases to make a correct diagnosis and to differentiate between those for patients requiring operative and nonoperative treatment. It is, therefore, important that in his training he should be exposed to patients with traumatic vascular injuries, vasospastic diseases, collagen diseases, venous and arterial thromboembolism, as well as those with atherosclerotic problems.

Good training by competent vascular surgeons is currently available through a variety of mechanisms: (1) within some general surgical residency programs; (2) within some thoracic and cardiovascular surgical training programs; (3) through vascular surgery fellowships.

General Surgery Residency Programs

There are several general surgical residency programs in which the residents completing their residency have had a broad experience with vascular diseases and have participated in a significant number of vascular operations. With the cooperation of The Conference Committee in Graduate Education, the operative experience records of 83 residents who completed their general surgical residency in 22 selected university hospitals or large clinics were reviewed. There were 19 residents from 9 different institutions who considered that they assumed enough responsibility that they were the surgeon on 40 or more vascular reconstructions during their training. Communications with some of these residents from our institution, as well as others, indicate that all or a

significant amount of their practice is vascular surgery, for which they were well prepared. It is still important that general surgeons obtain sufficient training to recognize and treat vascular diseases that require emergency operations and to be able to recognize more complicated problems which require referral to surgeons competent in vascular surgery with larger case loads and more adequate ancillary services. There are centers that by altering the residents' schedules and centralizing their vascular operations on one service, could provide training for general surgeons as well as a more optimal case load of vascular reconstructions for senior residents who wish to be vascular surgeons.[11]

Thoracic and Cardiovascular Residency Programs

There are thoracic and cardiovascular residency programs in which surgeons can obtain training in vascular surgery as well as thoracic and cardiac surgery. The associations of these specialties are logical and of historical interest. An example of the close association is the pioneering efforts of Gross in repairing coarctation of the aorta with end-to-end anastomoses, and the insertion of homografts. The principles and techniques established in these aortic reconstructions were important in the development of both cardiac and vascular surgery. There are thoracic programs such as those of Julian, Ochsner, and Parsonnet which make a definite point of providing training in vascular surgery. The advantages of a continued interest in both cardiac and vascular surgery can also be observed in the programs of DeBakey and Cooley. Julian has even recommended that some "thoracic programs be encouraged to develop strong sections in vascular surgery and that the qualifications required of a vascular surgeon lean heavily upon his being a product of such a program." On the other hand, there are centers that have found that the explosive increase in the numbers of cardiac operations, particularly with the advent of aortocoronary bypass grafts, has left little time for vascular operations and training. The operative experience forms, which are used for evaluation of training programs by the Residencies Review Committee for Thoracic Surgery and are also submitted by the resident with his application for board examinations, are also of some interest. The form does not include a specific request for the resident to list his experience with any of the operations within the realm of vascular surgery as earlier defined in this article. The American Board of Thoracic Surgery has recently stated that "before acceptance for examination, candidates will have completed acceptable training in all facets of thoracic surgery including thoracic, cardiac, and thoracic vascular surgery." The wording of its new certificates indicates that it is a "certificate in the specialty of thoracic and cardiac surgery."[10] On the other hand, on the basis of past concurrent growth with vascular surgery, the many contributions of thoracic surgeons to vascular surgical operations, and the involvement of many practicing thoracic surgeons in vascular surgery, it is important to know that the board is interested in and participating in discussions of certification of competence in vascular surgery.

Vascular Surgery Fellowships

There are various types of vascular surgery fellowships available. Some of them are in the form of 1 year fellowships in the basic problems

of vascular diseases which provide an opportunity to perform laboratory and clinical research in the detection, management, and prevention of peripheral vascular diseases, such as that offered by Hume. Another type of fellowship offers a combination of research and limited clinical experience with a group of vascular surgeons, such as the 2 year program offered by Cranley. The 1 year fellowships offered by Bergan, Dale, Thompson, and Wylie are primarily clinical and include experience in the preoperative work-up, angiography, operation, and postoperative care. These later fellowships are in hospitals where residents in general surgery also participate in the care of the patient. The fellowships of Linton and now Darling, are more like a preceptorship with the trainee participating in the office as well as the hospital care of private patients.

Junior and Senior Experience

Many competent surgeons who are now devoting a significant amount of their time to the treatment of vascular diseases have obtained their training through general surgical residencies, thoracic residencies, fellowship programs, or a combination of them. The understanding that good training can be obtained in different ways was considered by the Committee on Vascular Surgery of the Inter-Society Commission for Heart Disease Resources when they described the essentials of a vascular surgery training program.[11] They felt that ideally the training should consist of at least 12 months junior experience and 12 months senior experience.

Junior experience could be obtained during the junior years of many general surgical or thoracic and cardiovascular surgical residency programs. It might be obtained completely on a vascular service or on a general surgical service where vascular cases were concentrated. It is more likely that it would be obtained on rotations through several different services. It was felt that no more than 3 months' credit should be given for time spent on a strictly cardiac and thoracic service; no more than 3 months' credit for vascular radiology experience; and no more than 6 months' credit for time devoted to vascular research. Although not mentioned in the report, it is obvious that similar experience might be obtained in some vascular fellowships.

The 12 months of senior experience could be obtained in a special vascular surgical program or fellowship but might also be obtained in the final 2 years of a general surgical or thoracic residency program. It was felt advisable that at least 12 months' time be spent on a vascular service or general surgical service where vascular cases were concentrated but that 3 months' credit might be given for time spent on a cardiac and thoracic service. The Committee did recommend the number of operations that should be performed as first assistant surgeon with major responsibility, or surgeon, during the 12 months of senior experience, as follows: (1) arterial reconstructions and embolectomies: minimum, 40; optimum, 70; (2) major venous (portocaval shunts, interruptions, thrombectomies, and grafts), venous (saphenous ligations and strippings), amputations, sympathectomies, thoracic outlet decompressions, and arteriovenous shunts: minimum, 30; optimum, 30; total procedures performed: minimum, 70; optimum, 100. All of the problems of attempting to decide

how many operations a surgeon must perform to make him competent were considered by the Committee. The minimum figures were considered not to be optimal but are realistic figures based on numbers performed by senior residents who were considered to be competent vascular surgeons by the author and other consultants.

An important concept of what constitutes senior operative experience was discussed in the report. Although it would be preferable that the trainee be the surgeon, it is recognized that there are many training programs where the first assistant is given enough responsibility at the operating room table to provide equivalent experience. In fact, training experience as first assistant surgeon with major responsibility may well be superior to major responsibility without adequate supervision, even at the senior level of training. This changing concept of what constitutes senior experience was more bluntly expressed by Moore when he stated "The Halstedian residency system is dead."[8] He pointed out that the Halsteds residency system was based on the presence of a financially underprivileged group of patients. With the more recent realization that all segments of our population were entitled to the same level of medical care and the concomitant growth of health insurance, Medicare, and Medicaid, the chief resident's service suffered. Much of senior training, therefore, must be the shared responsibility of the surgeon and first assistant, both in decision making and in the performance of the operation.

CERTIFICATION

At the present time there is no recognized method for assessing the qualifications of a surgeon desiring to perform vascular operations. As most surgical specialties have matured they have established boards to assess qualifications and certify competency. Since 1916 ten separate surgical specialties have established boards, the last being colon and rectal surgery in 1949. Since then there has been considerable concern about the proliferation of specialties and boards. Clagett has asked for the recognition of the universality of surgery and admonished us not to "weaken our profession by division of surgery into weak branches and twigs."[2] Wagensteen has expressed the fear that the weakening of general surgery by increasing specialization will result in "master surgeons of many operations, but few to continue infusing into our discipline the life-giving spirit of research."[13] In 1970 a central board to represent all specialty boards, including the surgical specialties, was established and called the American Board of Medical Specialties. One of the purposes of the board was "to deal with applications for approval at proposed new specialty boards, subsidiary boards, new types of certification, modifications of existing types of certification and related matters."[6] Apparently this group has declared a moratorium on the development of new boards.[5] On the other hand, the American Board of Surgery has recently established a policy allowing for the awarding of certificates of special competence. This policy, in essence, allows for the development of sub-boards under the same board that certifies general surgeons. It was to the American Board of Surgery, therefore, that the Society for Vascular Surgery

and the North American Chapter of the International Cardiovascular Society addressed their resolution recommending and endorsing a certification method for recognition of special competence in vascular surgery under the aegis of the American Board of Surgery. The same resolution was considered by the Board of Regents of the American College of Surgeons in October of 1972. The Regents approved, in principle, the establishment of such a certificate for individuals who have been fully trained in general surgery and recommended that this be kept under the aegis of the American Board of Surgery. The resolution and a preliminary proposal were tentatively accepted at the June 13th, 1973 meeting of the American Board of Surgery. An ad hoc committee of surgeons has been asked to provide the board with a more concise definition of a vascular training program. If and when the American Board of Surgery accepts the proposal, a letter of intent would be filed with the American Board of Medical Specialties and after a layover period of 1 year, the American Board of Medical Specialties would act upon the proposal.

In summary, excellence in vascular surgery is dependent on special training. General surgery training programs can provide a sound basis or even complete training but in many instances an additional training period of 1 or even 2 years may be required for an adequate experience. It is hoped that in the future individuals who gain competency in vascular surgery can be recognized by special certification by the American Board of Surgery.

APPENDIX

The following is a list of training programs in vascular surgery which have been brought to the attention of the author. No attempt has been made by the author to investigate or judge the quality of the training programs listed. There is no way of stating whether the programs listed will or will not be considered qualified training programs if and when a Certification in Competence in Vascular Surgery is established.

Baird, R. J., M.D., Suite 301, East Block, 389 Bathurst St., Toronto, 2B, Ontario, Canada
Toronto Western Hospital
Bergan, J., M.D., 251 E. Chicago Ave., Chicago, Illinois 60611
Northwestern University Hospital
Blakemore, W. S., M.D., 19th & Lombard Sts., Philadelphia, Pennsylvania 19146
Graduate Hospital, University of Pennsylvania

Clauss, R. H., M.D., 1249 Fifth Ave., New York, New York 10029
New York Medical College Hospitals
Connolly, J. E., M.D., University of California at Irvine, Irvine, California 92664
University of California at Irvine
Cranley, J. J., Jr., M.D., 311 Howell Ave., Cincinnati, Ohio 45220
Good Samaritan Hospital
Crawford, E. S., M.D., Baylor University College of Medicine, Houston, Texas 77025
Baylor University College of Medicine
Curi, E., M.D., Newark, New Jersey
United Hospitals Medical Center

Dale, W. A., M.D., 2000 Church St., Nashville, Tennessee 37203
St. Thomas Hospital

Darling, R. C., M.D., Massachusetts General Hospital, Boston, Massachusetts 02114
Massachusetts General Hospital
DeBakey, M., M.D., Baylor University, Houston, Texas 77025
Baylor University
Deterling, R. A., M.D., 117 Harrison Ave., Boston, Massachusetts 02111
New England Medical Center
Diethrick, E. B., M.D., 350 W. Thomas Rd., Phoenix, Arizona 85001
Arizona Heart Institute
Downs, A. R., M.D., Winnipeg General Hospital, Winnipeg, Manitoba, Canada
Winnipeg General Hospital

Ehrenhaft, J. L., M.D., 325 Beldon, Iowa City, Iowa 52240
University Hospital
Evans, W. E., M.D., 410 W. 10th Ave., Columbus, Ohio 43210
University Hospital

Fogarty, T. J., M.D., 770 Welch Rd., Suite 201, Palo Alto, California 94304
Stanford Hospital
Foster, J. H., M.D., 3701 Whitland Ave., Nashville, Tennessee 37203
Vanderbilt University
Fry, William J., M.D., 1405 E. Ann St., Ann Arbor, Michigan 48103
University of Michigan Hospital

Hardy, J. D., M.D., 2500 N. State St., Jackson, Mississippi 39216
University of Mississippi, V. A. Hospital
Harrington, O. B., M.D., Baptist Hospital, Memphis, Tennessee
Baptist Hospital
Harrison, R. C., M.D., Vancouver General Hospital, Vancouver, British Columbia, Canada
Vancouver General Hospital
Hume, M., M.D., 170 Morton St., Jamaica Plain, Massachusetts 02130
Humphries, A. W., M.D., 2020 E. 93rd St., Cleveland, Ohio 44103
Cleveland Clinic

Imparato, A. M., M.D., 566 First Ave., New York, New York 10016
New York University, Bellevue Hospital
Inahara, T., M.D., St. Vincent's Hospital, Portland, Oregon
St. Vincent's Hospital

Jacobson, J. H. II, M.D., 1176 Fifth Ave., New York, New York 10029
Mt. Sinai Hospital
Jordan, P., Jr., M.D., William Beaumont Hospital, Detroit, Michigan
William Beaumont Hospital
Julian, O. C., M.D., 1725 W. Harrison, Chicago, Illinois 60612

Kaupp, H. A., Jr., M.D., 2004 Allen St., Allentown, Pennsylvania 18104
Allentown Hospital
Key, J. A., M.D., 8th Floor, University Wing, 101 College St., Toronto, Ontario, Canada
Toronto General Hospital

Linton, R. R., M.D., 1180 Beacon St., Brookline, Massachusetts 02146

McAllister, F. F., M.D., 180 Fort Washington Ave., New York, New York 10032
Presbyterian Hospital
Movius, H. J. II, M.D., Suite 706, 17 E. 8th St., Long Beach, California 90815
St. Mary's Hospital

Navarre, J. R., M.D., St. Vincent's Hospital, Toledo, Ohio
St. Vincent's Hospital

Nelson, R. M., M.D., Surgical Research Lab., LDS Hospital, Salt Lake City, Utah 84103
University of Utah, Latter-Day Saints Affiliated Hospitals
Noon, G. P., M.D., 1200 Moursund, Houston, Texas 77025
Methodist Hospital

Ochsner, J. L., M.D., 1514 Jefferson Highway, New Orleans, Louisiana 70121
Ochsner Clinic

Parsonnet, V., M.D., 116 Milburn Ave., Milburn, New Jersey 07041
Beth Israel Hospital
Pate, J. W., M.D., 858 Madison Ave., Memphis, Tennessee 38103
Baptist Hospital
Perdue, G. D., Jr., M.D., Emory University Hospital, Atlanta, Georgia 30322
Emory University Hospital
Pratt, G. H., M.D., 58 E. 66th St., New York, New York 10021
St. Vincent and St. Clare Hospital

Rich, N. M., M.D., Walter Reed Medical Center, Washington, D.C. 20012
Walter Reed Medical Center
Rob, Charles G., M.D., 260 Crittenden Blvd., Rochester, New York 14642
University of Rochester
Roberts, B., M.D., 3400 Spruce St., Philadelphia, Pennsylvania 19104
Hospital of the University of Pennsylvania

Sabiston, D. C., Jr., M.D., Duke University Hospital, Durham, North Carolina 27706
Duke University Hospital
Sauvage, L. R., M.D., 1001 Broadway, Suite 320, Seattle, Washington 98122
Providence Hospital
Stemmer, E. A., M.D., 5901 E. 7th St., Long Beach, California 90801
Veterans Hospital
Stiles, Q. R., M.D., 1136 W. 6th St., Los Angeles, California 90017
Good Samaritan Hospital
Szilagyi, D. E., M.D., 2799 W. Grand Blvd., Detroit, Michigan 48202
Henry Ford Hospital

Thompson, J. E., M.D., 3434 Swiss Ave., Suite 400, Dallas, Texas 75204
Baylor Hospital
Tsapagoas, M. J., M.D., Veterans Administration Hospital, Albany, New York
Veterans Administration Hospital
Tyson, R. R., M.D., Temple University Hospital, Philadelphia, Pennsylvania
Temple University Hospital

Wylie, E. J., M.D., 350 Parnasus Ave., San Francisco, California 94117
Moffitt Hospital, University of California San Francisco.

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Evaluation of the Patient for Vascular Surgery

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The patient with vascular disease presents with a spectrum of symptoms and signs which reflect the location and extent of the underlying process. At the time of the patient's initial visit, it is possible to decide on clinical grounds alone whether or not the patient will require more extensive evaluation. The extent to which the patient is subjected to further studies is largely dependent upon the attitude and experience of the vascular surgeon as well as the available hospital facilities. It is clear that at the present time most patients with occlusive arterial disease are subjected to operation based upon the history, physical examination, and appropriate angiography. With aneurysmal disease, arteriography may or may not be utilized depending upon the location of the diseased arterial segment.

While the standard approach to occlusive vascular disease is satisfactory, physiologic testing procedures are being implemented on a larger scale to complement the information obtained from the routine workup. This has come about for a variety of reasons, the principal one being the need for objective information indicating the manner in which the disease affects the functional status of the patient. Furthermore, it is now evident that many of these physiologic studies can be repeated as often as desired and the results used to indicate the degree of the success of the operative procedure as well as to detect new disease as it develops.

In this article, I will attempt to present a balanced appraisal of the current available methods of evaluation and how the results of these studies can be useful in evaluating the patient for vascular surgery.

ESTABLISHING THE DIAGNOSIS

The patient with arteriosclerosis obliterans (ASO) presents with either intermittent claudication and/or symptoms at rest (ischemic rest pain, ulcers, gangrene). The clinical presentation is well known and will

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not be detailed here except to emphasize those conditions which can mimic and be confused with true intermittent claudication. In recent years more attention has been devoted to the "pseudoclaudication syndromes" which are secondary to neurospinal disease.[9, 15] There are several features of this syndrome which distinguish it from true intermittent claudication, some of which are as follows: (1) The exercise-pain-rest cycle is often not typical or constant. (2) The symptoms often include numbness, tingling, weakness, incoordination, and clumsiness. (3) Patients with pseudoclaudication may have to sit down or lie down for relief. (4) The time required for pain relief may be excessive (often 20 to 30 minutes). It is very important that this entity be kept in mind. The author has seen several patients who, upon careful examination, were properly categorized as having neurospinal disease as the basis for their leg complaints.

When the patient complains of instep claudication, the possibility of thromboangiitis obliterans (TAO) must be considered.[12, 22] While it can occur with ASO, it is distinctly unusual and is nearly always present when TAO is the underlying problem.

The physical examination is useful in validating the patient's symptoms. It is important to look for the following: (1) absent or diminished pulses distal to most proximal level of involvement; (2) the presence of bruits or thrills arising from sites of narrowing; (3) the disappearance of foot pulses (when palpable) after exercise; (4) the appearance of a pale foot immediately after exercise; and (5) careful examination for the presence of an aneurysm. The performance of a careful history and physical examination is usually sufficient to establish the probable diagnosis and the most proximal level of disease involvement.

PHYSIOLOGIC STUDIES

Arterial narrowing and occlusion force the blood to follow the alternate pathways (collaterals) which are high resistance conduits and result in the development of an abnormal pressure gradient across the area of involvement. With early disease or single segment occlusions and good collaterals, the first change is simply a decrease in the systolic pressure distally. If occlusions are present at more than one level, the resistances of the collateral beds which are in series are additive, producing an even further decrease in the distal intraarterial pressure.[17] As the disease involves more major vessels and critical collateral pathways at either the stem (origin) or reentry sites, distal pressure may be very low and flow will become less and less pulsatile. Although systolic pressure in particular nearly always shows a decrease distal to areas of arterial obstruction, most studies of skin and muscle blood flow have shown that the resting blood flow level is nearly always normal. The abnormalities in flow that occur secondary to the arterial obstruction can be brought out by stressing the circulation either by exercise or 5 minutes of arterial occlusion (the reactive hyperemia test). However, at the level of the foot and digits, perfusion must be extremely variable, thus explaining the occurrence of rest pain and tissue necrosis confined to rather limited areas. The avail-

able study methods that have proved useful in estimating the extent of the physiologic derangements will now be considered.

Pressure Measurements

It is a common and accepted practice to assess the presence and severity of systemic arterial hypertension by the measurement of arm blood pressure. In the case of acute or chronic arterial occlusion, the problem of regional hypotension develops, whose course with and without treatment can be assessed if the technique for measuring pressure is available and applicable to the situation at hand. Since it is usually not possible to measure pressure distal to areas of narrowing or occlusion by using either Korotkoff sounds or return of palpable pulses, it is necessary to use some other indirect method. A variety of techniques have been developed which are applicable to both the upper and lower extremities and include the ultrasonic velocity detector,[20, 26] the mercury strain gauge plethysmograph,[19] and capacitance pulse pickups.[2] All of these instruments are used simply as sensors which indicate the point at which blood flow has been restored to the limb when a pneumatic cuff has been deflated from above the level of the regional blood pressure perfusing the limb.

The sensor is usually placed either over the digits, foot (plethysmograph), or the dorsalis pedis or posterior tibial artery (velocity detector). The systolic blood pressure can be estimated at the level of the ankle, below knee, above knee and upper thigh and then compared with that recorded from the upper arm. As has been reported, the systolic pressure at the ankle is normally equal to or higher than the brachial systolic pressure.[23] With arterial occlusion at one or more levels, the systolic pressure recorded at the ankle accurately reflects the extent of the proximal resistance and resulting pressure drop. Since the ankle pressure is also a reflection of the systemic arterial pressure, it is important to express the measurements as the arm/ankle pressure ratio (normal is $\leqq 1$). This ratio permits a comparison of the measurements on a day-to-day or month-to-month basis even though the systemic blood pressure may vary over a wide range.

The value of this simple test is that it accurately reflects the extent of the resistance to flow and can be used to assess the course of the occlusive arterial disease with and without therapy. For example, if new disease develops which increases or adds new areas of resistance to flow, the ankle pressure index will increase even further. Conversely, successful arterial reconstruction will return the index toward normalcy, the extent of which is determined by the nature of the procedure and its efficacy in bypassing or removing the areas of abnormally high resistance.

The sensitivity of the ankle systolic pressure test can be increased by making the measurements before and after exercise to the point of claudication.[3, 21] In contrast to the normal situation where moderate treadmill exercise (2 m.p.h. on a 12 per cent grade) results in no fall in the ankle systolic pressure, the patient with intermittent claudication will sustain a decrease in ankle pressure with a delayed return to baseline levels (the period of postexercise hyperemia). The value of this simple test is that each patient has his own response which is dependent upon the location and extent of involvement and represents his circulatory

response to stress. The addition of stress also tests the ability of the collateral circulation to respond to the increased flow requirements brought on by exercising.

Flow Measurement

The ultimate factor which determines whether or not a patient develops symptoms with arterial occlusion is the amount of blood flow delivered to the tissue in question and its requirements. Unfortunately blood flow is difficult to measure, particularly using noninvasive techniques, and the information is not as useful for clinical purposes as it might seem. Furthermore, we are unable to measure flow during exercise, the time during which the critical flow changes are taking place that are responsible for the symptoms.

In spite of the difficulties, measurement of regional limb blood flow using either plethysmography or isotope clearance techniques has provided valuable insight into some of the physiologic changes that accompany chronic arterial occlusion. Measuring techniques currently available can be classified by the anatomic segments to which they can be applied. Skin and muscle blood flow at the calf level are best estimated by using either volume or strain gauge plethysmography.[6, 21] Digit blood flow, which is primarily skin, can be measured using plethysmography. Muscle blood flow, independent of skin, can be estimated only by isotope clearance methods such as ^{133}Xenon.

Regardless of the measurement technique used, it is now accepted that resting flow determinations are of little value since the results are usually in the normal range even with rather extensive multilevel disease. The most valuable measurements are made immediately postexercise or after 5 minutes of inflow ischemia. Under these circumstances, limb blood flow requirements are greatly increased, the situation in which the functional abnormalities become most apparent. An extensive amount of work has been done in this area which can best be summarized as follows: (1) Normally peak muscle blood flow is reached within less than a minute after exercise, rapidly decreasing to reach preexercise levels within 1 to 2 minutes following a moderate workload. (2) In the patient with claudication, the peak level of muscle flow achievable via the collaterals is often lower than normal (< 15 ml. per 100 ml. tissue per min.) and the recovery time is greatly prolonged.[8] (3) Skin blood flow, particularly in the foot and digits, will decrease often remarkably in the patient with claudication as flow is directed to the exercising muscle.

Combined measurements of ankle systolic pressure and calf blood flow relating them to the level and extent of the occlusive disease have provided valuable information regarding the pathophysiology of intermittent claudication. The ankle systolic pressure response has been shown to be a reliable indicator of the flow change and is useful in estimating the severity of the occlusive disease.[21] Furthermore, since the pressure measurements are easier to perform, it is now clear that flow need not be measured to characterize the nature of the physiologic derangement accurately. Since pressures can be measured even with limited facilities and instrumentation, they are preferred as the routine determination in evaluating and following patients with ASO.

ARTERIOGRAPHY

This examination is nearly always done prior to reconstructive arterial surgery. This test provides the roadmap for the surgeon but, as is well known, often tends to underestimate the amount of disease present. Since this procedure is generally done in a single plane only, plaques present on the posterior wall of the arteries are often missed. Recognition of this deficiency is extremely important and should not lead the surgeon to select the operative procedure to be employed by the appearance of the arteriogram alone. This is particularly true where endarterectomy is being considered. The final decision as to whether a bypass graft or endarterectomy is most suitable is made with the vessel exposed so that the exact extent of the disease can be more accurately assessed.

CHOICE OF PROCEDURE

With single segment occlusions, the procedure to be employed will depend upon the extent of the involvement and the preference of the surgeon. Endarterectomy has its greatest application with disease limited to short segments, particularly in the aortoiliac area. Bypass grafts are more commonly applied when the disease is more extensive. The condition of the patient with regard to coexisting disease obviously also plays a major role in the decision as to which procedure should be employed.

With multilevel disease, it is now common practice to correct the proximal disease first and await the results. When multilevel disease leads to the development of rest pain, ulceration, or limited gangrene, successful bypass of the first group of collaterals is usually sufficient. When claudication alone is the problem, the end result is not as certain.[18] Sufficient experience has been gained by physiologic studies to show that in some patients, proximal reconstruction alone can often double or triple the walking distance. Unfortunately, there is a small group of patients who, when carefully studied after operation by exercise testing, will not have significant improvement even though the more proximal lesion has been successfully corrected. This means that the second series of collaterals has such high resistance that even providing a higher pressure at their origin does not result in an appreciable increase in flow during the stress of exercise.

INTRAOPERATIVE MANAGEMENT

When direct arterial surgery is being performed for occlusive disease the immediate result will be dependent upon the nature of the outflow vessels (the "runoff") and the technical success achieved in bypassing or removing the obstructing lesion. With a single segment occlusion such as in the aorta, iliac or femoral vessels, the procedure should restore the distal pressure and flow to normal levels. With multilevel disease and proximal reconstruction alone, distal pressure and flow should be improved but not restored to normalcy.

The problem for the vascular surgeon is to immediately assess the success of the reconstruction in the operative field and its pressure and flow contribution to the distal runoff vessels. It is now clear that return of pulsations in the host vessel or the bypass graft is not necessarily synonymous with immediate success. All vascular surgeons have had the experience of observing "normal" pulses in the operative field with no increase in distal flow due either to a thrombus, embolus, or intimal flap.

There are several methods of assessing the immediate result each of which provides the surgeon with some information and reassurance of the operative result. The first and most widely available technique is the operative arteriogram where dye is introduced often proximal to the reconstruction and films are taken of not only the corrected segment but those vessels supplying the lower limb. Some surgeons have advocated its routine use when an endarterectomy has been performed to be certain there are no residual debris or intimal flaps which may compromise the final result.

Other methods used to evaluate the results in the operating room have used either pressure or flow measurements as the index of success or failure. Pressures can be measured distally (at the ankle) or in the operative area.[7] Pressures measured in the vicinity of the reconstruction itself are not as useful since they provide little or no information about the flow contribution to the distal vessels. Furthermore, finding a normal pressure in the graft or endarterectomized segment does not insure a good result since a complete arterial occlusion can be present distal to the site of measurement. If a low pressure is noted, however, this is indicative of either a decrease in systemic pressure or of problems at the proximal end of the reconstruction.

Pressures at the ankle can be estimated by using the ultrasonic velocity detector or the mercury strain gauge plethysmograph. In practice the pressures are measured at both ankles prior to beginning the procedure and compared with that recorded at the arm. After the procedure, the pressures are remeasured at which time it is also possible to assess the effects of digital occlusion of the bypass graft or endarterectomized segment on the recorded pressures. In theory bypassing a single segment occlusion should restore the ankle pressure to normal. With proximal reconstruction in the presence of distal disease the ankle pressure should be expected to increase but not to reach normal levels.

In practice several points must be kept in mind: (1) Restoration of the ankle pressure to normal levels is reassurance that under the conditions of measurement there are no significant areas of narrowing or occlusion. (2) If the distal pressure increases, but not to normal levels with single segment disease, it may be a reflection of a distal embolus or production of an intimal flap. If an epidural or spinal anesthetic has been used or if the blood volume is decreased, the ankle pressure may not come up to normal levels immediately, even though the reconstruction has been completely successful. (3) With multilevel disease, an increase in the ankle pressure is certain evidence that a flow increase has taken place but it is always difficult to predict the level of pressure increase to be expected under these circumstances.

Estimation of flow either by direct measurements using the elec-

tromagnetic flowmeter or the ultrasonic velocity detector in the operative field can be very helpful in assessing the immediate results.[1, 4, 5, 10] With the electromagnetic flowmeter blood flow can be measured before and after the reconstruction with or without the use of intraarterial papaverine. The same goal may be accomplished using the ultrasonic velocity detector with the major difference being that the surgeon listens to the change in flow velocity that has accompanied the reconstruction. With a sterile velocity probe, it is possible to examine the flow contributions of the vessel proximal to the reconstruction, the reconstruction itself, and those vessels in the vicinity that serve as the major outflow conduits. Obstruction to flow is immediately recognized by either no signal or the "water-hammer" pulse observed in cases of acute arterial obstruction. Our experience has been similar to that published by Keitzer et al.[10] who have used the velocity detector extensively in this area. Of the 93 patients monitored, eight (8.6 per cent) required 14 reoperations. There were 49 other patients who were not monitored with 15 (30.6 per cent) requiring 31 reoperations.

POSTOPERATIVE EVALUATION

In-Hospital

By paying close attention to the reconstruction and the distal vessels by pressure and flow estimations and arteriography at the time of operation, the incidence of immediate or in-hospital complications has been greatly reduced. It has been our distinct impression that most problems which lead to early failure occur in the operating room and are missed. By relying more heavily on intraoperative measurements, the incidence of reoperations required in the early postoperative period should be kept at a minimum.

If pedal pulses return and are normal this is reassuring, but we have come to rely more on measurements of the ankle blood pressures in order to remove observer bias. Furthermore, in patients with multilevel disease, pedal pulses would not be expected to return and are thus not available as an index of the continued success of the procedure.

Long-Term Evaluation

After discharge from the hospital, the long-term success can be estimated in terms of (1) patient response (relief of symptoms); (2) estimation of the hemodynamic effectiveness of the procedure; (3) the fate of the reconstruction itself; and (4) the occurrence of new disease in proximity to or at sites removed from the reconstructed segment.

The patient's response to therapy is obviously the most important and key element in evaluating the results of any therapeutic procedure. The problem with relying on this form of evaluation alone is that it is subjective and interpreted by the two people most intimately connected with the procedure – the patient and the surgeon. The patient's desire for improvement coincides with the surgeon's hope for a good result. The other major problem with subjective evaluation alone is that it is relatively insensitive. For example, the patient's estimate of changes in walking tol-

erance depends upon a variety of factors which are difficult to quantitate. This fact is clearly brought out when a more rigorous approach to postoperative assessment is carried out.

Physiologic studies probably play their greatest role in the long-term follow-up of patients who have undergone direct arterial surgery.[16] New disease at sites removed or problems in the reconstruction itself can be detected well before the patient or physician is alerted to the fact that changes are taking place. Furthermore, it permits the surgeon to separate the effects of disease progression from those secondary to problems within the reconstruction itself. How can this be done?

At each follow-up visit, the patient is questioned and the physical examination repeated. It is usually possible to tell at this point whether the reconstructed segment is open but it tells little else. The arm and ankle systolic pressures are remeasured and expressed as the arm/ankle pressure ratio. The patient is then exercised on the treadmill either to the point of claudication or to a full 5 minutes of walking at 2 m.p.h. on a 12 per cent grade. The postexercise ankle pressures are measured to assess the extent of the change and if they decrease the time required for the pressure to return to the pre-exercise level. If the resting arm/ankle pressure ratio has increased, new problems in that limb are a certainty. If the resting ratios are unchanged but the postexercise ankle pressure decrease is greater and the recovery time prolonged, the area(s) of new disease is significant only with the stress of exercise.

If problems have been discovered, a more diligent search is carried out to localize the area involved. This is done by remeasuring the segmental pressure gradients, looking for the site of the new pressure decrease and a careful survey with the ultrasonic velocity detector. The sites examined include the reconstruction itself, the external iliac, common femoral, superficial femoral, popliteal, and tibial arteries. Combining the pressure measurements with the velocity detector examination will permit localization of the problem in nearly all cases.

If the patient is symptomatic or if the new disease appears to be in a location which may jeopardize the reconstruction, repeat arteriography is often recommended. However, we have on numerous occasions simply followed the patient at more frequent intervals (monthly) to assess the rapidity with which the new disease is occurring. These repeated observations are very useful in estimating the extent to which the circulation to a limb is deteriorating. Furthermore, it is not at all uncommon to develop new disease which in large part can be ameliorated by improvement in collateral artery function which is important to document.

We have carried out such long-term studies of three groups of patients: aortic aneurysms; aortoiliac reconstructions; and femoropopliteal operations. The results of these studies can be summarized as follows:

Abdominal aortic aneurysms.[25] Sixty-one patients followed up to 12 years. In 44 per cent of the patients with ASO at the time of resection, disease progression occurred in 44 per cent of cases as compared to 17 per cent in the patients free of occlusive disease at the time of operation.

Aortoiliac reconstructions for ASO.[14] Sixty-three patients followed for an average of 4.7 years. The overall long-term failure rate of the reconstruction was 29 per cent. In terms of disease progression – patients with disease confined to the

aortoiliac segment only – 14 per cent developed new disease as compared to 38 per cent in those patients with disease at more than one level.

Femoropopliteal reconstructions.[13] Fifty-seven patients had 62 operations followed for an average of 3.3 years. Twenty-one per cent of the procedures failed with disease progression occurring in 34 per cent of the limbs studied. Of the 21 patients with disease progression, eight operations were required to return the patient back to a better functional status.

This brief summary of the results of our follow-up studies using physiologic methods is mentioned to emphasize that the techniques are not only practical for long-term studies but may be used to identify and separate reconstruction failure from disease progression. It has also been possible to avert failure of the reconstruction in some patients by early detection of the developing problems.

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Application of Ultrasound to Arterial and Venous Diagnosis

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It is in the best traditions of medicine and science that physicians have attempted to extend their senses in order to detect and measure physiologic alterations in blood flow and pressure. Just as the stethoscope increases auditory sensibility and the sphygmomanometer quantitates a palpable arterial pressure, ultrasound instrumentation increases physician ability to observe and so to diagnose blood flow abnormalities in arteries and veins.

Ultrasound waves as used in medicine may be classified by intensity. High intensity ultrasound (20 to 100 MHz) is destructive and may be used in therapy. Low intensity ultrasound (1 to 10 MHz), in contrast, is not harmful and is utilized in diagnosis to investigate physical properties of the tissues through which it passes without causing permanent change.

Application of ultrasound in medicine is recent. In 1960, Satomura and Kaneko, using an ultrasonic blood rheograph based on the principle of the Doppler effect, first described a noninvasive method of studying instantaneous changes in blood flow in human peripheral arteries.[8] Later, Strandness and Rushmer (1966) popularized transcutaneous flow detection to study peripheral vascular problems.[11] Until that time, diagnosis or objective assessment of limb ischemia was dependent upon clinical examination, angiography, or the cumbersome technique of plethysmography. Development of the continuous-wave or pulsed Doppler opened a new field for clinical investigation in patients with peripheral vascular disease.

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INSTRUMENTATION

Ultrasound consists of high-frequency sound waves beyond that portion of the sound spectrum audible to humans, usually above 20 KHz. They can be produced by electrically exciting a piezoelectric crystal. Such a crystal changes its dimensions under the influence of an electric field and will radiate energy in the form of sound waves. Similarly, a crystal can be used as an ultrasound detector when the dimensions of the crystal are changed by a sound beam striking it and a voltage change is developed which can be detected and amplified. Thus, the crystal can be used to emit sound beams and to detect a sound field.

Two types of ultrasound beams are currently used in diagnosis. These are the pulsed and the continuous-wave emission instruments.

Continuous-Wave Doppler (CWD)

There are now two commercially available types of Doppler ultrasound flow velocity meters utilizing continuous-wave emission. These are the nondirectional and the directional velocity meters. The transmission frequency of these flow velocity detectors varies from 2 to 10 MHz (megacycles per second). A frequency of 5 MHz gives a deep penetration and a comparatively broad beam, especially suited to deep blood vessel flow in the vena cava or iliac veins. An operating frequency of 10 MHz is less penetrating but permits a sharper focus and is ideal for blood velocity detection in arteries and veins of limbs and digits.

In principle, an ultrasound beam of 5 or 10 megacycles per second emitting from a ceramic crystal excited by an electrical oscillator is coupled to the skin with a gel and then passed through an underlying blood vessel. Ultrasound reflected from red cells in the blood vessel is shifted in frequency by an amount proportional to the flow velocity of the red cells. The back-scattered sound is detected by another crystal mounted adjacent to the transmitting crystal. This back-scattered sound

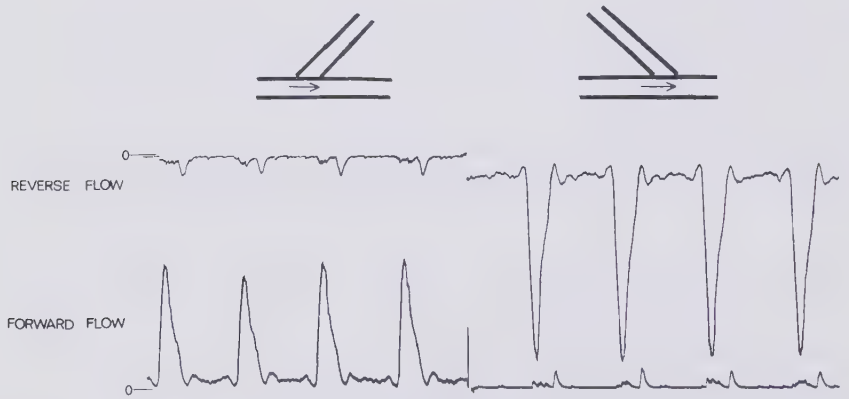

Figure 1. Demonstration of the directional capability of the transcutaneous directional Doppler instrument using dual output presentation. A positive Doppler shift is seen when the probe is pointed upstream. A mirror image is obtained when the probe is reversed. Note the switch of flow signals in the reverse and forward flow channels.

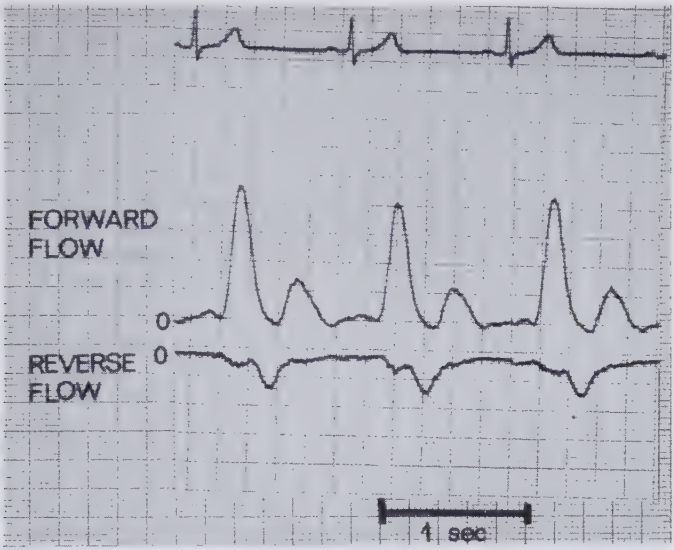

Figure 2. Doppler flow velocity pattern recorded by the dual output method from the common femoral artery in a normal subject. Reverse flow appeared to occur when forward flow was beginning to decelerate.

is mixed with the transmitted frequency to produce a signal within audible range. The pitch of this audio frequency signal is proportional to the velocity of blood flow within the vessel under study. A high velocity of blood movement causes a higher-pitched sound than a low velocity movement. The audible output from this instrument may be used to drive a loudspeaker or earphones.

The development of a directional Doppler by McCleod represented a major advance in transcutaneous flow detection.[5] Directional flow detection is accomplished by introducing a phase-shift network prior to flow detection, permitting the identification and separation of positive and negative Doppler shifts. A 90° phase-shift network is used and, depending on the direction of blood flow (forward or reverse), the phase angle either adds to or subtracts from the Doppler shift. Two methods of analog presentation are available in directional flowmeters. The composite wave form of the integrated forward and reverse flow components may be seen on a single channel recorder (see Fig. 5), or the forward and reverse flows may be displayed separately on a two channel recorder (Figs. 1 and 2).

Pulsed Doppler

Pulsed ultrasound operates on the same echo-ranging principle as naval sonar systems. With the pulsed Doppler, a pulsed beam is produced by electrically exciting a piezoelectric crystal for a short time. Following the periodic short burst of ultrasound, the instrument examines for Doppler shift by sampling the signals reflected from discrete points along the ultrasonic beam at different times. Since the transmitted signal is pulsed rather than continuous, it is possible to sample blood flow from a small volume of tissue at any point along the sound beam.

Of interest to vascular surgeons is the development of a method of visualizing the arterial lumen utilizing this technique. As reported by Mozersky[6] and Fish,[2] the pulsed Doppler not only charts the flow velocity profile, but also allows transcutaneous determination of the cross-section of the artery. When cross-sectional dimension and velocity are known, blood flow can be calculated.

METHODS

Auscultation

Auscultation of the heart using a stethoscope is an established routine in study of cardiac function. The Doppler ultrasound method has made it possible to study sound in peripheral arteries and veins in a similar manner. Audible signals can be obtained by placing the flow probe over an artery. Normal arterial sounds consist of first, second, and third sounds; the pitch rises abruptly to a high peak during systole and falls during diastole. Arterial sounds are recognized by the changes in velocity during each cardiac cycle. Venous sounds, in contrast, are respiratory-dependent.

In patients with occlusive or stenotic lesions of the proximal arteries and in whom pedal pulses are not palpable, flow signals can usually be obtained over the posterior tibial or dorsalis pedis arteries. Abnormal sounds are heard which result from collateral flow and these are of low pitch. The second and third sounds are absent. A flow signal detected immediately below a stenotic segment senses high velocity flow and is characterized by a high pitched sound superimposed upon continuous low frequency sound. This indicates the presence of turbulent flow.

Unlike cardiac dependent arterial sounds, normal venous sound is cyclic with respiration. The sound resembles the noise produced by a wind storm. On breath-holding or Valsalva maneuver, flow ceases and no sound is heard. With sudden release of intrathoracic pressure or inspiration, there is rapid venous flow from the limb and a loud roaring sound is heard.

Venous flow can be detected distal to an occlusion or proximal to a site of thrombosis. In patients with total proximal venous occlusion, sound recorded distal to the site of blockage does not show respiratory modulation. It is continuous and rumbling in character and is unmodified by breath-holding or the Valsalva maneuver. Diagnosis of venous occlusion may be made by detecting an increase of venous flow velocity by external compression. In the presence of a normal patent deep venous system, a surge of blood can be heard in the common femoral vein when the calf muscle is squeezed by the examiner. This sound, termed "augmented sound," was first described by Siegal and his colleagues.[9] Similar maneuvers can be used over the popliteal and calf veins. By placing the flow probe behind the knee, popliteal vein flow can be studied. Absence or marked diminution of induced signals suggests occlusion between the site of pressure and the flow probe.

Flow Velocity Wave-Forms

Audible sounds can be converted into analog output and displayed graphically. Normal arterial flow velocity is triphasic in pattern, corresponding to audible sounds (see Fig. 5). The major deflection represents forward flow during systole; the second deflection is caused by reversed flow of a low frequency during diastole; the third signal represents the return of forward flow. In nondirectional instruments, both forward and reverse flows are expressed as a positive deflection on the wave-form tracing. With the directional Doppler, the second deflection is placed below the "zero" flow line.

Abnormal sound such as that recorded distal to an occlusion is monophasic when displayed graphically. This flow velocity pattern results from collateral vessel flow and is characterized by a slow acceleration during systole, delayed deceleration, low systolic peak velocity, and absence of distinct second and third deflections.

The flow velocity pattern obtained immediately below a stenotic lesion where high velocity flow is present is manifested by a rapid rise and fall of systolic flow with absent second and third deflections. The flow pattern is irregular whenever there is a palpable thrill.

Venous flow velocity patterns are distinguished by the presence of a slow clear respiratory cycle. Breath-holding and the Valsalva maneuver cause a flat wave form to appear. The blockage of effluent blood terminates with resumption of respiration and a high flow rate is usually seen. In patients with total proximal venous occlusion, respiratory waves are not transmitted and no change in flow rate is induced after the Valsalva maneuver.

Systolic Pressure Measurement

Audible signals can be detected by using the flow probe as a stethoscope over the posterior tibial or dorsalis pedis artery. If the blood pressure cuff is applied around the ankle above the malleolus, ankle systolic pressures are recorded at the pressure at which flow signals return during deflation of the cuff. Segmental limb pressure can be recorded by placing the cuff around the calf or the thigh.

In normal subjects, the ankle pressure when measured in a supine position is equal to or higher than that in the upper extremity. A pressure index or percentage of brachial pressure, as suggested by Winsor,[13] is obtained by comparing the systolic pressure in the lower extremity with that in the upper extremity. The pressure index is useful in grading the degree of ischemia. In normal individuals, the pressure index in all instances is greater than 1.0 but in patients with angiographic evidence of occlusion, the pressure index is always less than 1.0. In patients with intermittent claudication, the average pressure index is 0.59, whereas in rest pain or impending gangrene the pressure index ranges from 0.26 to 0.05 respectively.[14]

The simplicity of pressure measurement allows instantaneous determination of the effect of exercise following a standard treadmill test. Moderate exercise causes a slight increase in ankle blood pressure. Ces-

sation of muscle activity allows ankle pressure to return rapidly to normal. These physiologic responses become pathologic in patients with arterial occlusion. A profound drop in ankle pressure is usually found with exercise.

Direction of Blood Flow

The development of the directional Doppler allows demonstration of the direction of blood flow under physiologic conditions. A positive or negative Doppler shift can be obtained, depending upon the position of the probe, which is aimed either toward or away from the flow stream (see Fig. 1). Reversal of blood flow, as in arterial steal phenomena, can be detected easily.

Flow Volume

If the cross-sectional area of an artery is determined through use of such methods as the echo track[7] or the pulsed ultrasonic velocity detector (PUVD),[6] flow volume can be calculated transcutaneously. In determination of flow volume, the sonogram method is more accurate than the commercially available frequency voltage converting technique.[7] The mean velocity is obtained by subtracting the reversed flow area from the total area to give the net frequency shift for forward flow alone.

Assuming flow is parabolic, mean flow velocity can be considered as one half of the peak frequency. Once the mean frequency shift is established, it is possible to calculate mean velocity from the following formula:

$$V = \frac{\Delta f \times C}{2f\ (\text{Cos}\ \Theta)}$$

where V = velocity
f = frequency of incident sound beam
C = velocity of sound in tissue (1.56×10^5)
Θ = angle of incident sound beam

Mean flow is then calculated from:

$$Q = AV$$

with Q = flow
A = cross-section area
$\overline{V}$ = mean velocity

Identification of Turbulent Flow

Turbulent flow is defined as "an irregular condition of flow in which the various quantities show a random variation with time and space coordinates."[1] Since the Doppler method depends upon the fact that in laminar flow the red cells, which reflect the ultrasound beam, are moving in a parabolic form with the cells in the center of the stream moving faster than those at the periphery, the result is a spectrum of velocities rather than a single velocity. In the presence of turbulent flow, as when recording is done distal to a stenotic lesion, the red cells are flowing in different directions and at different velocities; therefore, by analyzing the frequency spectrum of Doppler flow signals, it is possible to detect the

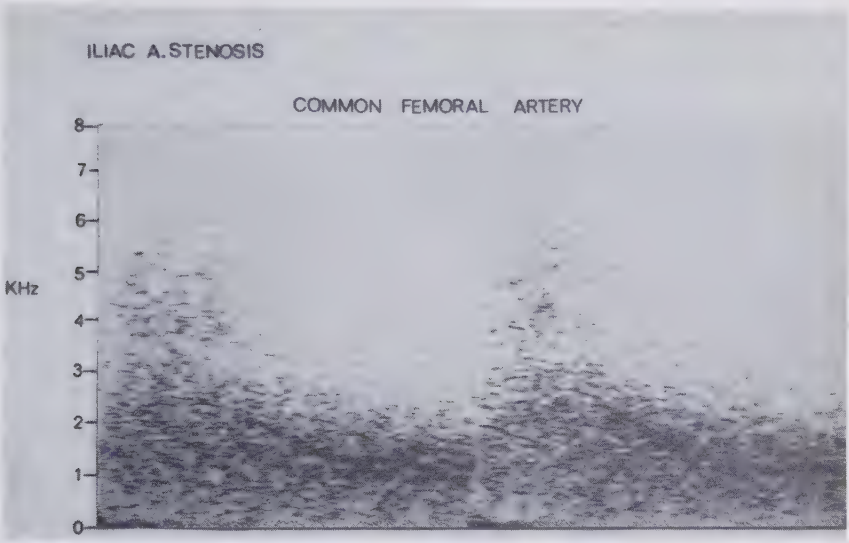

Figure 3. Flow signals recorded from the common femoral artery in a patient with iliac artery stenosis. Turbulent flow displayed by sonogram is characterized by the lack of pulsatility and increase of both high and low frequency components.

presence of turbulent flow in intact arteries in man. Both low and high frequency components are increased if turbulent flow is present (Fig. 3).

CLINICAL APPLICATIONS

Occlusive Arterial Disease

LOWER EXTREMITY. The most common cause of occlusive disease affecting arteries supplying the lower extremity is arteriosclerosis obliterans. Despite the presence of arterial occlusion and impalpable distal artery pulsations, flow signals can be obtained over the posterior tibial or dorsalis pedis arteries. Objective quantitation of the degree of ischemia can be achieved by noting the presence or absence of pulsatile flow, measuring ankle blood pressure, determining the ankle-brachial pressure index, and identifying turbulent flow.

Objective assessment of patient symptomatology can be done by ultrasound techniques. Determining the ankle pressure level and noting the pressure index readily identifies those patients who have arterial intermittent claudication and differentiates them from those patients who have leg pain on exercise from other causes. Such differentiation is difficult in patients with intact peripheral pulses. Of 34 patients examined in the Blood Flow Laboratory who had exercise leg pain and palpable pedal pulses, all had abnormal flow velocity patterns in the posterior tibial and dorsalis pedis arteries. In all of these patients, the ankle pressure was less than the brachial systolic pressure, and exercise provoked

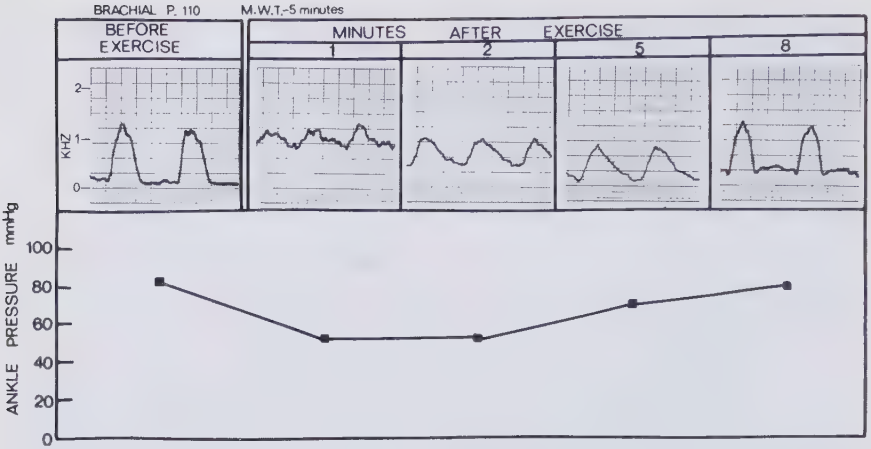

Figure 4. Simultaneous recording of posterior tibial artery flow velocity patterns and ankle pressure before and after exercise in a patient with femoral artery occlusion. Both flow and pressure decreased after treadmill exercise.

a marked decrease in both pressure and flow recorded from the posterior tibial artery (Fig. 4).

Ankle pressure supplements angiographic information on the status of flow distal to arterial occlusions. A pressure index ranging from 0.40 to 0.50 is usually accompanied by good distal runoff. Even in the presence of angiographic demonstration of poor distal runoff, a reconstructable popliteal artery can be assured if the ankle pressure is above 20 mm. Hg. When flow or pressure from pedal arteries is not detectable and there is angiographic evidence of poor runoff, attempted limb salvage angioplasty is unlikely to succeed.

When arterial reconstruction is not feasible, amputation is often necessary. Determination of the level of amputation is facilitated by measurement of thigh systolic pressure. In 36 patients facing inevitable amputation, thigh systolic pressure above 80 mm. Hg assured successful below the knee amputation. In ten patients with a thigh systolic pressure below 80 mm. Hg, nine failed to achieve primary healing of below knee amputation and all required a more proximal revision.

The importance of recognizing the presence of stenosis in the iliac artery prior to superficial femoral artery reconstruction is well appreciated by vascular surgeons. Such proximal stenosis is common and its physiologic importance must be considered. Identification of turbulent flow in the common femoral artery after exercise is comparatively simple using the Doppler technique. This gives objective information about the hemodynamic significance of iliac artery stenosis. In normal subjects, the pulsatile flow pattern following moderate exercise on the treadmill is characterized by an increase in systolic velocity and absence of reverse flow (Fig. 5). In the presence of critical stenosis of the aortoiliac segment, turbulent flow is seen in the common femoral artery after exercise. This is manifested by continuous flow with marked velocity increase in both high and low frequency components; simultaneous ankle pressure meas-

urement reveals a profound concomitant drop in ankle systolic pressure.

The directional Doppler also permits study of flow phenomena within femoral artery aneurysms. Reversed flow along the wall of the aneurysm has been noted and this finding accounts for gradual expansion of the aneurysm (Fig. 6).

DETERMINATION OF PATENCY OF A RECONSTRUCTED ARTERY. Restoration of arterial pulses at the ankle level is desirable after arterial reconstruction. Unfortunately, complex and multiple arterial occlusions produce the severe ischemia which most often requires correction. Therefore, even after successful arterial grafting, ankle pulses often remain absent. The Doppler ultrasound method provides a tool for use at the bedside and is of particular value with patients in whom there is a residual lesion in addition to that undergoing correction by surgery and in whom return of the pedal pulse is unlikely. Graft patency can be easily monitored during the immediate postoperative period (24 hours) by frequent measurement of ankle and brachial systolic pressures. If the pressure index fails to increase after operation despite adequate blood volume replacement during the first 6 to 12 hour period, occlusion by thrombosis is likely (Fig. 7).

UPPER EXTREMITY. Arteries accessible for transcutaneous flow examination are the subclavian, axillary, brachial, radial, ulnar, and all digital arteries. The diagnosis of occlusion of the subclavian artery can easily be made by noting monophasic flow pattern in the brachial artery and its branches.

In patients exhibiting digital ischemia or Raynaud's phenomenon, this method is particularly applicable. A change of flow velocity in the brachial artery during various thoracic outlet maneuvers establishes the diagnosis of thoracic outlet syndrome. The presence of digital artery thrombus alerts the physician to causes such as scleroderma or rheumatoid arthritis. The noninvasive nature of this technique appears to make it an ideal screening procedure prior to definitive arteriography.

CEREBRAL VASCULAR INSUFFICIENCY. The Doppler ultrasound technique offers new approaches to the assessment of cerebral ischemia. In addition to establishing the presence of atheroma in the carotid artery by

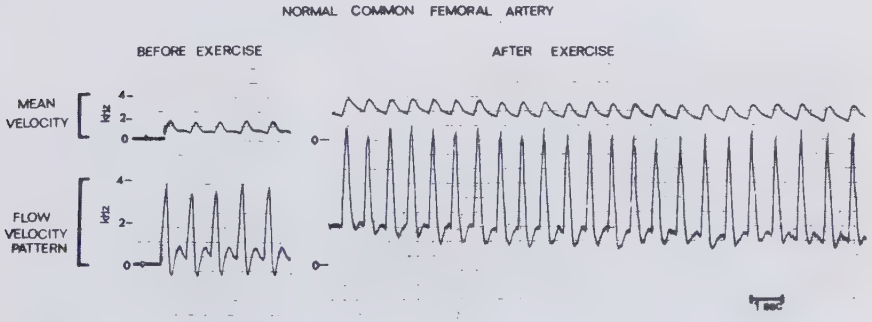

Figure 5. Pulsatile flow and mean flow velocity recorded from the common femoral artery in a normal subject before and after treadmill exercise. Disappearance of the reverse flow component together with an increase in mean flow velocity are indicative of vasodilation, a normal occurrence after moderate exercise.

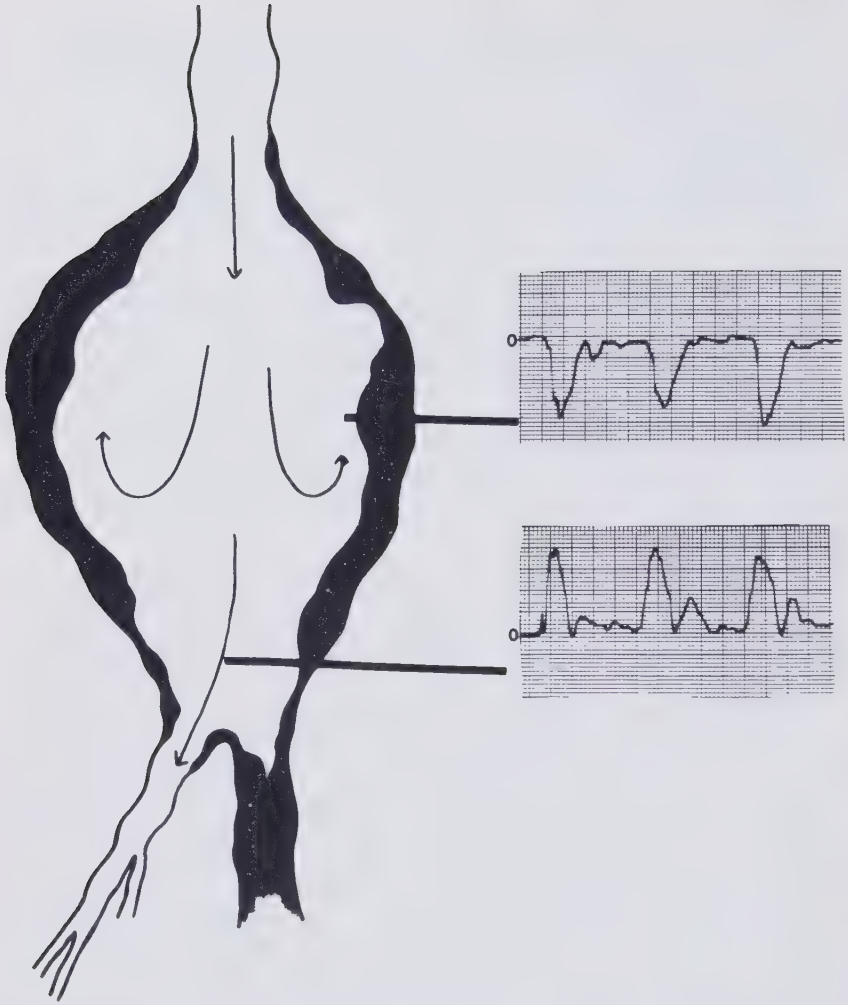

Figure 6. Variation of the direction of flow velocity when recorded in the periphery and in the center of an expansile aneurysm of the common femoral artery. Reverse flow is seen in the wall of the aneurysm.

detection of turbulent flow, the supraorbital and vertebral arteries can be examined by this method. The supraorbital artery, a terminal branch of the internal carotid artery, serves as a convenient outlet for studying internal carotid artery flow. In the presence of total occlusion of the internal carotid artery, reverse flow is often demonstrated in the supraorbital artery (Fig. 8). Change in the direction of blood flow while the superficial temporal artery is momentarily compressed further substantiates reversed extracranial blood supply to the brain when there is total occlusion of the internal carotid artery.

There may be no bruit when the internal carotid artery is completely occluded. In this instance, detection of abnormal flow pattern and direc-

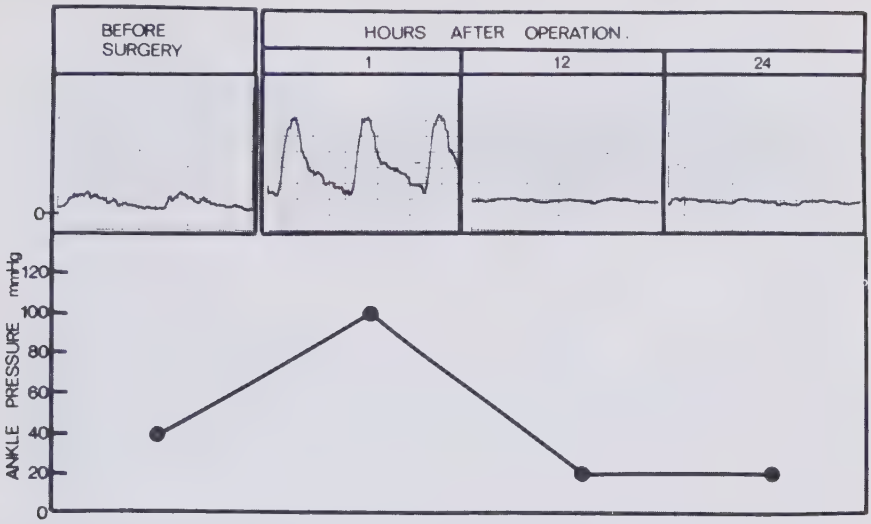

Figure 7. Monitoring of the flow velocity pattern and ankle systolic pressure in a patient who underwent femoropopliteal artery reconstruction. After initial success, occlusion of the graft was seen at 12 hours postoperatively.

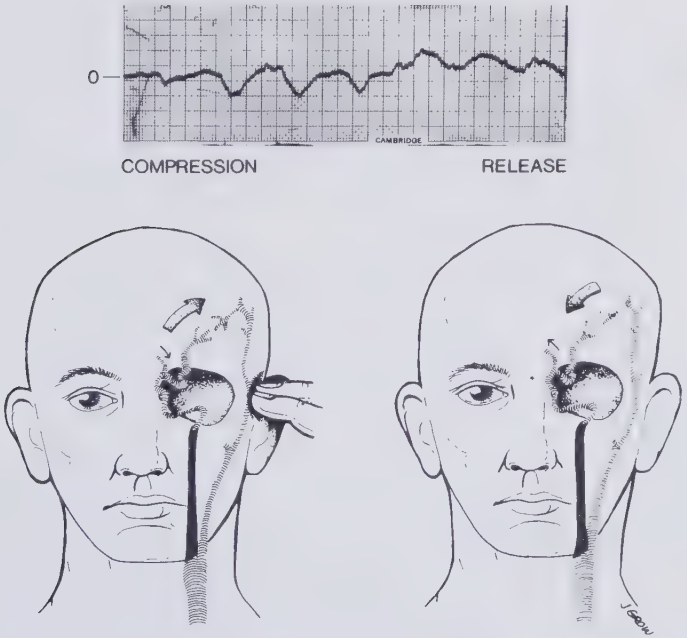

Figure 8. In a patient with complete occlusion of the internal carotid artery, extracranial flow to the brain can be achieved through the external carotid via anastomoses with the supraorbital artery (right). Reverse of flow direction can be demonstrated by directional flow recording of the supraorbital artery while the superficial temporal artery is being compressed. This finding is diagnostic for complete occlusion of the internal carotid artery even when no bruit is present.

Figure 9. Venous flow signals recorded in a communicating vein. Bi-direction of flow is demonstrated in a patient with valve incompetency.

tion of flow in the supraorbital artery aids in establishment of a diagnosis. Assessment of supraorbital artery flow is also helpful in bilateral carotid artery stenosis or occlusion. Manual compression of the superficial temporal artery and observation of the effect on the ipsilateral supraorbital artery flow enables more adequate assessment of the source of ischemic symptoms, thus permitting a better surgical approach.

"Subclavian steal," manifested angiographically by retrograde filling of the vertebral artery, can be readily diagnosed by demonstration of reverse flow using the directional Doppler. The presence of abnormal flow patterns and pressure in the brachial artery, accompanied by reversed flow in the vertebral artery following hyperemia induced by exercise or cuff occlusion, confirms the diagnosis instantly.

Acute and Chronic Venous Disorders

As indicated above, normal venous flow velocity is cyclic with respiration; in venous thrombosis, the respiratory effect is absent. Deep veins accessible for examination are the iliac, common femoral vein above and below the inguinal region, the femoral vein in the mid-thigh portion, and the popliteal vein behind the knee joint. Occasionally, the posterior tibial vein can be detected at the ankle.

In addition to recording changes in flow velocity elicited by respiratory movements, detection of augmented sound when the calf is squeezed provides a valuable index of patency. Although the diagnostic accuracy of the ultrasound method in detection of small clots in the calf veins is unreliable, the technique is useful in determination of the extension of thrombus into the popliteal vein via daily monitoring of the popliteal venous flow velocity.

The Doppler ultrasound method is also applicable in differentiation of the cause of chronic limb swelling. Detection of abnormal femoral venous flow velocity patterns suggests the presence of iliofemoral venous occlusion.[15] Valvular incompetency at the common femoral and popliteal level can be determined by the presence of negative flow while the abdominal pressure is increased. Similarly, incompetency of the saphenofemoral valve as the causative factor in development of varicose veins may be instantly detected by this method (Fig. 9). Chronic limb swelling due to valvular incompetency rather than thrombosis affecting major veins may be determined by this method.

COMMENT

Until McCleod (1967) and Kato and Izumi (1970) introduced the directional Doppler, most commercially available continuous wave Doppler instruments could not determine direction of blood flow. The ability to detect direction of blood flow has permitted several observations: (1) Flow reversal (negative flow), previously reported in the common femoral artery, can be demonstrated in distal arteries such as the posterior tibial and dorsalis pedis; reverse flow occurs during diastole, near the wall while there is still forward flow in the center core of the stream. (2) Negative flow disappears when proximal occlusion is present. (3) In the presence of low impedance vascular bed, as in hyperemia, the flow reversal component is always absent. (4) Reversed flow occurs along the wall of an expansile aneurysm. (5) Retrograde flow is recorded in the supraorbital artery when complete obstruction of the internal carotid artery is present; a similar finding is seen in the vertebral artery in patients with the subclavian steal syndrome.

The introduction of the transcutaneous flow detection method has greatly facilitated the measurement of systolic pressure in the lower limb. Pressure level recorded distal to an occlusion or stenosis reflects not only the degree of severity of the disease, but also the function of collateral vessels. The lower the pressure level, the greater the ischemia.[14] Ankle pressure level alone appears to be useful in confirming the clinical diagnosis of arterial occlusion.

Although angiographic classification of distal runoff bears close relationship to patency rate of the femoropopliteal graft, this technique has limitations. Angiography may be imprecise in delineating arteries distal to occlusions. Measurement of ankle pressure can supplement information on the adequacy of runoff distal to the popliteal segment. A pressure index greater than 0.45 invariably indicates good runoff. Even in the face of angiographic evidence of poor runoff, an ankle pressure greater than 20 mm. Hg, together with a pulsatile flow recorded in either the posterior tibial or dorsalis pedis artery, is a favorable indication for reconstructive surgery. When poor runoff is combined with absence of flow and pressure from the pedal arteries, a reconstructive procedure may be impossible.

When arterial reconstruction is not feasible, determination of the level of amputation may pose a problem. Optimal rehabilitation requires preservation of the knee joint. Conversely, attempted below-knee amputation in the presence of inadequate blood supply may necessitate re-amputation at a higher level, thus imposing additional risk to the patient. The simple method of measuring thigh pressure in determination of level of amputation has been investigated in the Blood Flow Laboratory. A thigh pressure above 80 mm. Hg assures success of below-knee amputation (Yao, unpublished data).

At the time of operation, Doppler ultrasound flow detection is employed to determine patency of the graft.[3] Upon completion of a femoropopliteal bypass graft, flow signals or recorded patterns proximal and distal to the anastomotic sites enable the vascular surgeon to assess graft patency in the operating room. An abrupt change in sound characteristics immediately distal to the site of anastomosis indicates technical diffi-

culty; the presence of turbulent flow is also an indication of technical error. Use of the directional flowmeter is even more informative. Blood flow direction is determined in the superficial femoral and popliteal arteries and in the saphenous vein graft. If the blood stream is flowing in the same direction in all three channels, success of the reconstruction is assured. Flow in the opposite direction in the femoral artery proximal to the anastomotic site usually indicates a defect in the anastomotic line. This finding is also seen when high impedance of the distal vascular bed is present due to diffuse arterial occlusion. Detection of technical error in the operating room permits its immediate correction; thus, graft failure can usually be corrected early. Keitzer and his colleagues have reported a significant reduction in reoperative rate in 142 patients on whom the Doppler flowmeter was used intraoperatively.

Postoperatively, Doppler ankle sphygmomanometry appears ideal for monitoring graft patency. This method of assessment is of particular value when femoral arterial reconstruction is done in the presence of diffuse involvement of distal arteries where the return of a pedal pulse is unlikely. It is also useful in patients with aortoiliac and superficial femoral artery occlusions where reconstruction is attempted only in the aortoiliac segment. Monitoring of the ankle systolic pressure and comparison with preoperative readings has proved to be a simple and objective method in determining patency of the reconstructed artery. Lack of improvement in ankle pressure within 12 hours after surgery invariably indicates graft thrombosis. Even when a pedal pulse is palpable after segmental reconstruction, the ankle pressure measurement furnishes a convenient way of monitoring graft patency during the immediate postoperative period. Whenever palpation of a pulse is in doubt, ankle pressure measurement will confirm or deny the presence of pedal pulses. Perhaps the chief advantage of this method occurs when a pulse thought to be present on palpation is actually nonexistent. To assume success when failure has occurred is a serious error in management. Technical faults detected early can often be corrected.

While occlusive arterial lesions are less common in the upper than in the lower extremities, the wide spectrum of neurologic and vascular disorders causing disability of the hand and fingers provides a diagnostic challenge to vascular surgeons. Detection of a change in flow velocity in the brachial artery during various thoracic outlet maneuvers differentiates symptoms due to arterial insufficiency from nerve compression. Discovery of digital artery occlusion often changes the diagnosis of Raynaud's disease to that of Raynaud's phenomenon and alerts the physician to search for an underlying disease such as rheumatoid arthritis or collagen disease as the cause of small artery thrombosis.

Diagnosis of extracranial arterial occlusive disease has heretofore been largely dependent upon auscultation of the carotid arteries in search of bruits. In general, presence of a bruit indicates that narrowing of the carotid artery has occurred. If the stenosis progresses, the bruit becomes inaudible. Therefore, absence of a bruit does not positively rule out occlusive arterial disease of the extracranial portion of the carotid artery. Assessment of supraorbital flow by ultrasound appears to provide information not obtainable by auscultation. In total occlusion of the internal

carotid artery where extracranial filling through the external carotid artery system has occurred, reversed blood flow direction in the supraorbital artery is observed by this technique. The supraorbital artery flow decreases or reverses while the superficial temporal artery is being compressed. This maneuver is unique and is also of value in the selection of the site of operation in patients with bilateral carotid artery occlusion.

Clinical evaluation of veins is more difficult than the assessment of arteries because there is no palpable venous pulse in the periphery. For the first time, a direct method of assessing venous hemodynamics at the bed side is available with the Doppler method. The application and value of the Doppler ultrasound technique in the diagnosis of lower extremity venous disorders has been described by several investigators.[9, 15] Accuracy of the technique ranged from 75 to 90 per cent as compared with venographic examination. Diagnostic values are higher in proximal major veins but the technique is probably not accurate in diagnosis of small clots affecting soleal veins or veins below the popliteal segment. Even though diagnostic value in this group of patients is limited, extension of thrombus from these small veins into the popliteal vein can be easily detected by this method. Thus, the method is not only a useful aid to diagnosis but is also a valuable means of following the progression of thrombosis.

While the continuous wave Doppler provides valuable clinical information, the pulsed Doppler instrument appears to hold great promise in vascular surgery. Unlike continuous wave instruments, pulsed ultrasound can provide information on flow velocity profile and transcutaneous measurement of arterial lumen size. The latter, termed "ultrasonic angiography," enables visualization of the lumen in anterior and posterior as well as lateral views.[6] The most useful feature of this technique is the ability to obtain the cross-sectional area of an artery. Such information is vital in the calculation of blood flow. Combined use of the two Doppler instruments will likely remove the reluctance of most clinicians to accept the continuous wave Doppler. In general, the clinician prefers to know the value of blood flow in ml. per min. rather than as it is expressed through Doppler calculations. Although flow volume is important, the study of pulsatile flow in arteries in terms of flow acceleration, velocity, and distal pressure, rather than mean flow alone, has recently been emphasized by several investigators.[3, 4, 12, 16] Apart from calculation of flow values, the ultrasound angiography technique is useful in following the progress of the atheroma. Preliminary application of this method has been reported by Strandness and his colleagues[10] and appears promising.

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Use of Radioisotopes in Assessment of Distal Blood Flow and Distal Blood Pressure in Arterial Insufficiency

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By recording the local clearance of radioactive isotopes in patients with arterial occlusive disease one can measure blood flow as well as blood pressure in muscles and skin distal of the occlusion. *Distal blood flow* is most conveniently measured as the muscle blood flow in the calf during hyperemia. This affords a sensitive diagnostic tool disclosing the presence of occlusive arterial lesions and it gives, in addition, some information regarding the site of this lesion. The *distal blood pressure* can also be measured in the muscles. But, we most often make these measurements in the skin on foot or leg because of the clinical usefulness of these measurements in treating impending or manifest ischemic skin lesions.

This article will thus be confined to a discussion of distal blood flow in the calf muscles and of distal blood pressure in the skin. The clinical value will be compared to that of other methods for measurement of flow and pressure below the arterial occlusion. As such physiologic measurements afford a key to the proper therapy, we shall also comment both on surgical and on medical therapy.

Local Clearance Method

Both radioisotopic techniques that will be described in this article are based on the local clearance method. The method was developed by Kety,[13] who in 1949 used the disappearance rate of radioactive sodium ($^{24}Na^{+}$) injected intramuscularly as a measure of muscle blood flow. Walder in 1958 demonstrated with this method that there is a reduced

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calf muscle blood flow during leg exercise in patients with arterial insufficiency of the leg.[25] He also showed that there is a much prolonged post-exercise hyperemic response. There have been described other radioisotopic methods for the evaluation of arterial insufficiency. The delayed arrival in the foot of intravenously injected radioactive tracers[6] and the distribution in the leg tissues of macroaggregated radio-iodinated serum albumin[12, 21] should in particular be mentioned. But these methods do not yield quantitative measures of blood flow or blood pressure in the distal tissues and have not found wider clinical application.

The local clearance method is based on the use of tracers that diffuse so freely between the tissue and the capillary blood that diffusion equilibrium is practically maintained regardless of the rate of blood flow. For these tracers it is the blood flow that is the limiting factor in the removal from a locally injected depot. Among such *flow limited* tracers are the radioactive inert gas ^{133}Xenon and 4–iodo–antipyrine labelled with ^{125}Iodine or with ^{131}Iodine. Small ionized tracer molecules such as ^{24}Sodium or ^{99m}Technetium (as pertechnetate) pass, on the other hand, more slowly across the capillary membrane. This means that during maximal hyperemia, at least in skeletal muscle, it is predominantly the capillary diffusion capacity (the permeability) which determines the rate of removal by the blood. Such tracers are thus mainly *diffusion limited* during hyperemia. But, at the very low flow that prevails when we measure distal blood pressure (see below) the small ions can be assumed to reach close to diffusion equilibrium between tissue and blood. Hence, in such measurements even these tracers are predominantly flow limited.

PRINCIPLE OF MUSCLE BLOOD FLOW

For measurement of muscle blood flow we use radioactive Xenon, about 0.1 ml. of sterile saline containing ^{133}Xe in physical solution (ca. 0.5 μc. per ml.) is injected intramuscularly.

The injection causes a moderate hyperemic reaction as evidenced by a slightly faster washout rate during the initial few minutes. This injection artifact is normally quite small and the measured blood flow relates quite closely to true muscle blood flow when measurement is made a few minutes after the injection.[23] No adverse local reactions have been encountered, not even in patients with poor peripheral circulation.

The only technical error of importance is the occasional deposition of the injectate in nonmuscle tissue, probably in adipose tissue. This error is encountered in about 1 per cent of injections and is recognized by a slowly decreasing clearance curve, which is unaffected by muscle exercise and which cannot be reproduced. Histamine is in some studies admixed to the ^{133}Xe-saline (20 μg. per 0.1 ml.) in order to produce local hyperemia.

The principle of measurement involves the recording of the disappearance of the ^{133}Xe from the local depot by using a scintillation detector coupled to a ratemeter. The calculation of the muscle blood flow (MBF) is made from the slope of the logarithmically recorded washout curve. This slope is proportional to the flow, a shallow slope corresponding to a slow

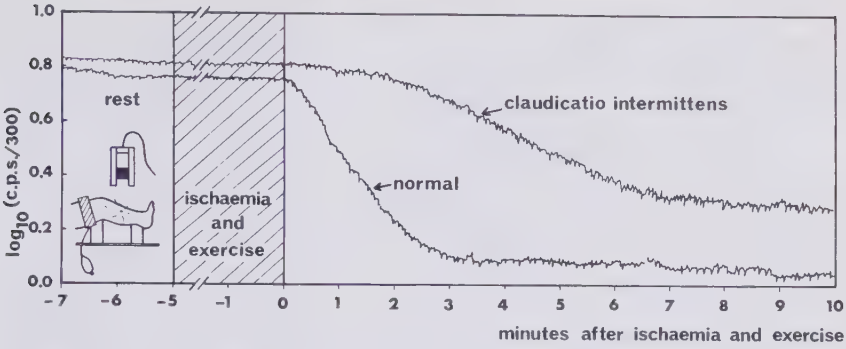

Figure 1. ^{133}Xe clearance curves from the anterior tibialis muscle in a normal subject and in a patient with obliterative arterial disease. Muscle hyperaemia is provoked by ischaemia and exercise. A delayed hyperaemia response is pathognomonic of the disease.

flow, a steep slope to a fast flow. The factor of proportionality is 161 if the flow, D, is measured as a fraction of 100 per minute:

$$MBF = 161 \times D \text{ ml. per 100 gm. per min.}^{1}$$

Measured by this method resting muscle blood flow is normally about 2 ml. per 100 gm. per min. in good agreement with estimates by plethysmographic techniques. During exercise the maximal muscle blood flow reaches levels of about 60 ml. per 100 gm. per min. in healthy adults.

Muscle Blood Flow in Occlusive Arterial Disease

We originally employed ischemia combined with exercise as a stimulus for eliciting muscle hyperemia.[15] In this ischemic exercise test the subject is studied in the supine position and the ^{133}Xe-saline is injected into the anterior tibial muscle, both sides being studied simultaneously (Fig. 1). After isotope injection the circulation to the legs is suddenly arrested by inflating cuffs placed above the knees. During the following 5 minutes the ischemia is maintained and the calf muscles are exercised to the point of fatigue and pain by ankle movements. When the cuff is suddenly released the clearance will in normal man increase to high levels corresponding to a muscle blood flow exceeding 35 ml. per 100 gm. per min. And this maximum is reached within about 20 seconds, maximally within 1 minute after cuff release. In patients with occlusive arterial disease the maximal flow is lower and it is reached more slowly. Some patients cannot cooperate in this test, failing to exercise the muscles to exhaustion. In such cases, one may use 5 minutes of ischemia alone, but then it is necessary to inject histamine along with the ^{133}Xe, as otherwise the accuracy of the test suffers.[17] These tests are positive in about 95 per cent of patients with arterial occlusion between the heart and the site of measurement. Failures are encountered in cases with good collaterals and occlusion quite close to the site of measurement. Because of these failures and because of the discomfort to the patient as well as the undesirability of employing occluding cuffs if it can be avoided, we now use only the ischemic exercise test in patients who cannot perform the walking test.

The walking test is made by injecting the ^{133}Xe-saline into the calf muscles (Fig. 2).[1, 2] Usually the medial head of the gastrocnemius muscle is studied. Light scintillation detectors are taped over the isotope depots, and after recording the resting clearance in the motionless standing position for about 2 minutes, the subject starts walking on a treadmill at a speed and elevation causing his typical symptoms to develop within about 2 minutes. Subjects not manifesting claudication are stopped after 3 to 5 minutes. After the walking test the patient stands motionless on the treadmill until the postexercise hyperemic phase has passed and a slow, fairly constant clearance rate approximating the pre-exercise resting value has become established. With this test the normal response consists of a muscle blood flow exceeding 10 ml per 100 gm. per min., and of a postexercise hyperemic period lasting less than 3 minutes. The patients usually have a lower blood flow during walking and they all have a delayed hyperemic response. The delay of maximal blood flow is a rough measure of the localization of the occlusion. Thus longer occlusions involving both the femoral and iliac arteries give a marked delay with the maximal postexercise hyperemia first being encountered with a delay of 4 minutes or more.

The walking test involves little discomfort and no risk to the patient. The entire test including calculations and evaluation requires 30 minutes and is performed by a nurse or a technician. It has proved reliable provided one really does inject that muscle giving pain. In cases of branch thrombosis below the knee, it gives diagnostic possibilities that cannot readily be obtained by calf plethysmography (Fig. 3).

Comments on the Routine Use of Muscle Blood Flow Measurements

The blood flow value obtained with the ^{133}Xe method is not very reproducible as it has a coefficient of variation of about 25 per cent. But the diagnostic criteria also include an evaluation of the shape of the ^{133}Xe washout curve. And using both flow and time course there is quite a wide and clearcut difference between normal and abnormal responses (Fig. 4). The variability in blood flow values does not invalidate the clinical routine use of the method.

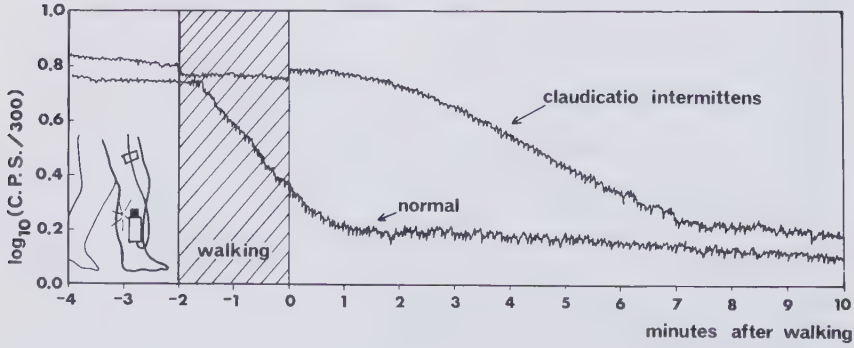

Figure 2. ^{133}Xe clearance curves from the gastrocnemius muscle in a normal subject and in a patient with obliterative arterial disease. The muscle hyperaemia is provoked by strenuous walking on a treadmill causing intermittent claudication in most patients with this complaint. The typical delayed hyperaemia in the patient corresponds to the pattern of the postischaemic-exercise test (Fig. 1).

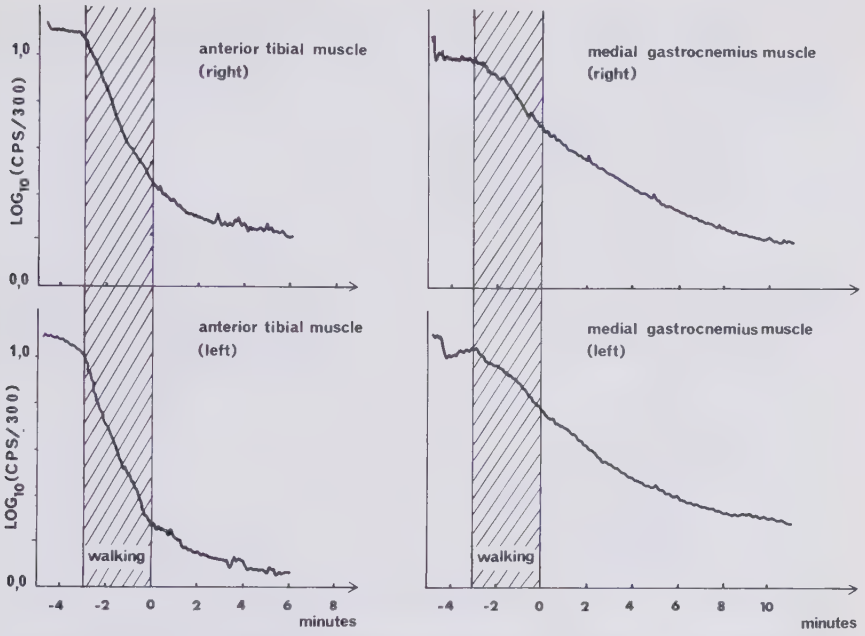

Figure 3. ^{133}Xe walking test performed in a patient with bilateral branch thrombosis of the posterior tibial artery. In the first test (left upper and lower panels) the isotope was injected into the anterior tibial muscle in both legs and the walking test was normal as can be seen from the figure.

In the second test (right upper and lower panels) the isotope was injected into the medial head of the gastrocnemius muscle and here the maximal blood flow was delayed and the resting-clearance was not established until 8 to 10 minutes later. The angiogram showed occlusion of the posterior tibial artery in both legs, whereas the anterior tibial artery was intact as was the proximal arterial tree.

The ^{133}Xe method can of course also be used for physiologic studies of muscle blood flow. Then a fairly large number of observations are often needed in order to reduce the random variation. We cannot here give an account of the various scientific results thus obtained. It may be appropriate, however, to tell that the spectacular improvement in walking tolerance that can be regularly obtained by exercise training of claudicants is associated with only a small increase in calf muscle blood flow during walking. Enzymatic adaptation[10] and *not* development of new collaterals is apparently the main mechanism of this therapy, which is so far the only form of conservative treatment of established value in patients with intermittent claudication.

Compared to calf plethysmography as applied to blood flow measurement, the ^{133}Xe method has the advantage of ease of performance: external compressing cuffs can usually be avoided and no standardization is needed (same normal and abnormal values have been found in various laboratories). In our experience the two blood flow methods are about equally effective (the advantage of ^{133}Xe in diagnosis of branch thrombosis has already been mentioned). In practically all cases they permit one to establish the diagnosis so that angiography can be reserved for use in situations when a surgical intervention must be considered. With the

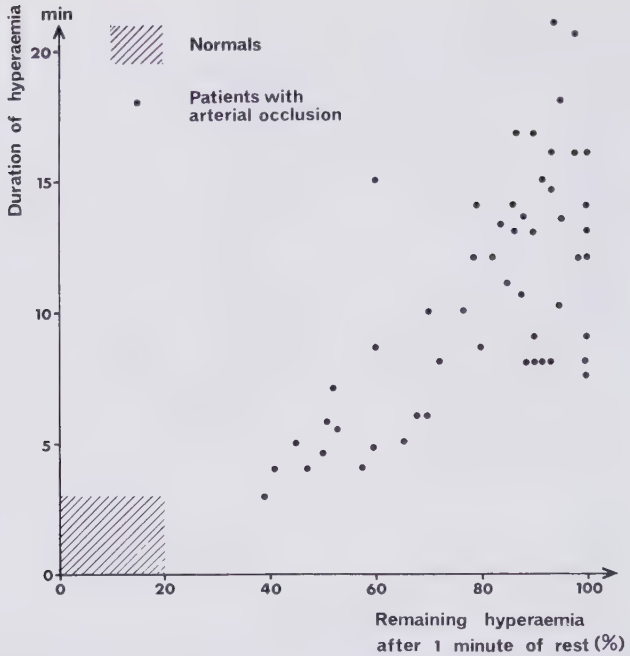

Figure 4. Timecourse of the ^{133}Xe washout curve from calf muscles: the relation between the duration of post-exercise hyperaemia and the percentage of hyperaemia after 1 minute of rest. Normal values (mean + 2 S.D.) are shown by the shaded area. The values from 52 calf muscles in 18 legs with arterial occlusion above the knee differ widely from the normals.[1]

benign spontaneous prognosis and with the symptomatic improvement obtained by walking exercise, reconstructive surgery is in our experience rarely indicated in elderly claudicants if they do not suffer in addition from severe distal hypotension at rest, as will be discussed below.

PRINCIPLE OF SKIN BLOOD PRESSURE

Assessment of the arterial blood pressure in a tissue as that external counterpressure which just suffices to arrest tissue blood flow was introduced in 1967.[7, 20] The flow cessation pressure measured by inflating a blood pressure cuff placed over a radioactive depot injected intramuscularly was found to be practically equal to the diastolic blood pressure in normal subjects. In patients with severe occlusive arterial disease this pressure is markedly reduced. This principle has been adapted here for measuring the local blood pressure in the skin.

We use 4-iodo-antipyrine labeled with ^{131}Iodine or ^{125}Iodine dissolved in sterile water at a concentration of about 0.2 μc. per ml. Histamine is always admixed to the radioactive solution, so that the injectate contains 50 μg. per 0.1 ml. This results in vasodilatation of the skin and minimizes the influence of temperature induced spontaneous variations in skin blood flow. ^{131}Iodide ($Na^{131}I$) can also be used. ^{133}Xe is not practical due to diffusion of this tracer into the subcutaneous adipose tissue in which it

has a high solubility and ^{99m}pertechnetate is washed out too slowly to allow exact interpretation of the clearance curve.

About 0.1 ml. of the tracer-histamine mixture is injected intradermally, so that a small papule is raised. The counterpressure is obtained by placing a conventional arm blood pressure cuff, so that it covers the depot. In order to measure the local pressure over the injected skin area even more precisely, we employ in addition a small, thin-walled plastic bag that is interposed between the depot and the blood pressure cuff. The bag is filled with a small amount of air (or water) and connected to a manometer (Fig. 5). It is this manometer and not that which one could connect to the outer cuff that we employ. The small plastic bag overlies the injected skin area smoothly (without wrinkles) even where the surface is irregular, as on the foot.

A scintillation detector is placed over the blood pressure cuff at a distance of about 10 cm. from the depot and the clearance is recorded semilogarithmically. When a constant clearance (a descending straight line) has been observed for about 3 minutes with no counterpressure applied, then this pressure is raised stepwise resulting in a stepwise decrease in isotope washout rate (Fig. 6). In order to determine the pressure at which the clearance stops it is necessary at the highest pressure steps (made at intervals of 5 mm. Hg) to observe the curve for about 5 minutes at each

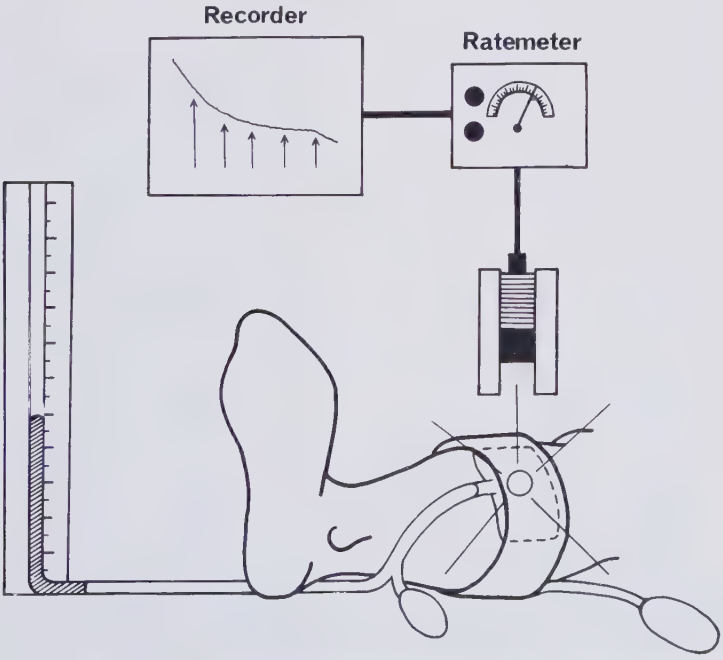

Figure 5. Measurement of skin blood pressure on the calf by the flow cessation counter pressure. The local clearance from an intradermal depot of ^{131}I-antipyrine mixed with histamine is registered by a scintillation detector coupled to a ratemeter, the output of which is recorded on a penwriter. Counterpressure to the depot is applied with an ordinary blood pressure cuff. A plastic bag containing a small amount of air is interposed between the cuff and the depot, and the pressure directly to the depot is measured by a manometer connected to this bag.

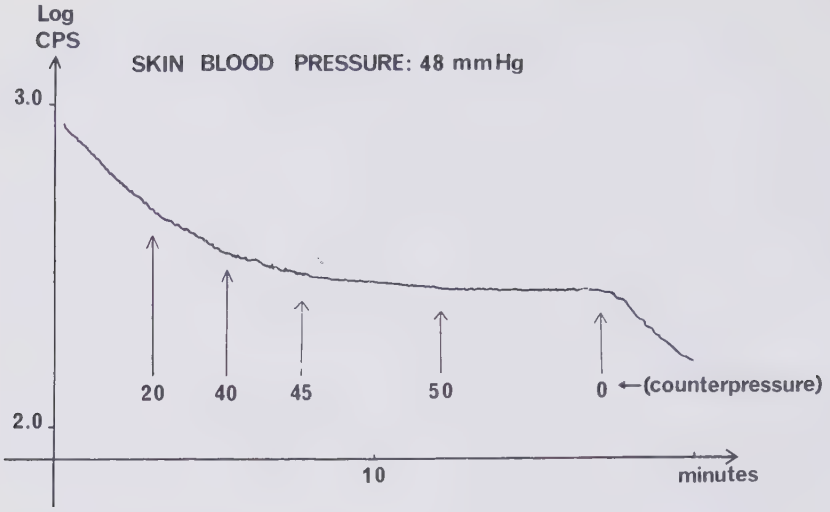

Figure 6. The local clearance from an intradermal depot of ^{131}I-antipyrine mixed with histamine. Stepwise increasing counter pressure results in a stepwise decrease in clearance until flow cessation occurs (flat curve).

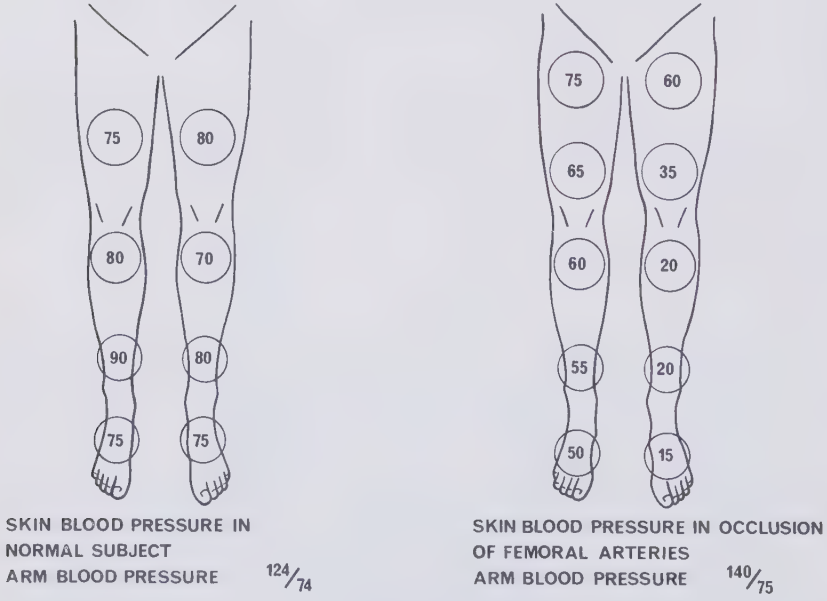

Figure 7. Skin blood pressure measured on both legs in a normal subject at various levels (*left*), and in a patient with vascular occlusive disease (*right*). In the patient's right leg there is an occlusion of the superficial femoral artery. In the left leg there is an occlusion of both the superficial and the deep femoral artery. Gangrene of the toes on the left leg was present. The skin pressure values are adjusted to one arm blood pressure level in each patient.

step. The skin blood pressure is taken to be the pressure midway between the highest pressure at which flow still can be discerned and the pressure level above this at which flow cessation is observed.

The most important source of error is the movement artifact. The leg must be kept immobile at a constant distance from the detector and this demands proper fixation of the leg. We use sandbags. Another source of error which is related to the movement artifact is seen when the skin is edematous. Then the external compression moving the fluid will cause the skin to move away from the detector. In case of edema the swelling should be squeezed away by applying a pressure of about 80 mm. Hg for 2 to 3 minutes prior to the study.

The room temperature should be "thermoneutral" (ca. 25° C.), so that the patient feels comfortable with light clothing and the skin temperature is warm (ca. 28 to 32° C). Arm blood pressure should always be taken simultaneously. We block the thyroid by 0.5 gm. of potassium iodide before injecting iodine containing tracers.

Skin Blood Pressure in Occlusive Arterial Disease

In normal man the external pressure on the skin which results in flow cessation lies close to the diastolic blood pressure regardless of the site measured from (Fig. 7). By external compression of a tissue the situation created is equivalent to a decrease of the arterial blood pressure. The observation in normal man implies, therefore, that when the *effective* local diastolic blood pressure is zero, then flow also stops.

Why this is so we do not know. The relation between flow cessation in the skin and diastolic blood pressure can probably be disrupted. Extreme bradycardia or marked tachycardia might do so. Severe hypertension or other conditions with marked pulse pressure might also cause the flow cessation pressure to be higher than the diastolic one. But in personal studies of patients with essential hypertension we have so far not encountered such cases.

It follows from the above considerations that in patients with occlusive arterial disease where the pulse pressure in the distal vascular bed is decreased, the skin blood pressure is a measure of the local diastolic blood pressure. This pressure is often normal in patients with claudication, while in patients with chronic ischemic skin lesions or rest pain the local pressure is invariably below 20 to 30 mm. Hg. In such low pressure areas skin lesions heal badly or not at all. This observation we consider of decisive clinical importance, as is illustrated by a series of major leg amputations recently studied in our clinic: 2 to 8 weeks after surgery skin blood pressure was measured on the anterolateral side of the stump 10 cm. from the wound (Fig. 8). Of the 51 cases reamputation at a more proximal level because of ischemic skin necrosis became necessary in 8 cases. The skin blood pressure at the level of the amputation was low in all 8 cases. If we divide the material into legs with skin pressures below 20 mm. Hg, between 20 and 40 mm. Hg, and above 40 mm. Hg, the result obtained was that (1) 5 out of 5 amputations with a skin blood pressure below 20 mm. Hg failed because of ischemic necrosis; (2) 3 out of 12 amputations with a skin blood pressure between 20 and 40 mm. Hg failed

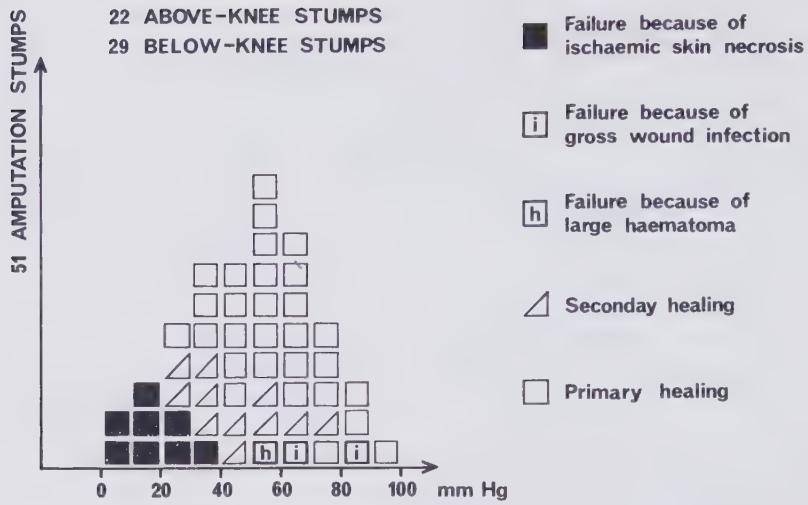

Figure 8. Skin blood pressure values measured 2 to 8 weeks postoperatively in 51 amputation stumps (29 below knee and 22 above knee). Ischaemic skin necrosis invariably caused reamputation when the skin blood pressure was below 20 mm. Hg. Between 20 and 39 mm. Hg skin necrosis resulted in failure or slow secondary wound healing in most cases. Among 34 stumps with a skin blood pressure of 40 mm. Hg or more there were three failures, due to wound infection or haematoma, but no failures because of skin necrosis.

because of ischemic necrosis; (3) none of 34 amputations with a skin blood pressure above 40 mm. Hg failed because of ischemic necrosis.

Secondary wound healing occurred in 11 subjects. It was our clinical impression that this complication was in many cases related to poor skin circulation especially where the skin sutures had been placed. This impression gains support by the skin blood pressure measurements showing many such cases in the 20 to 40 mm. Hg group.

It shall not be claimed that an adequate skin perfusion pressure is a guarantee against wound failure. There were 3 failures at high pressures, 2 severe infections and 1 large haematoma compromising the surgery. But, if the pressure is very low, then it appears that a large wound (the amputation) cannot heal. This is amply supported by our experience concerning the healing of traumatic or "spontaneous" skin lesions in patients with occlusive arterial disease: if the skin blood pressure is below approximately 20 mm. Hg, healing will not occur; in the pressure range 20 to 40 mm. Hg slow healing will usually occur; if the pressure is higher, then circulation is not a problem.

In the amputation material presented we gave the postoperative pressure because it corresponds to the pressure when healing must occur. The clinically important problem is, however, the *preoperative* assessment of the skin blood pressure. In most cases this pressure corresponds closely to the postoperative one, so that correct prognostic inferences could be drawn (Fig. 9). In low pressure regions the trauma of operation and bandage pressure may cause skin necrosis of extensive areas in which the pressure decreases to zero. But in other cases a moderate pressure rise was found postoperatively, and hence the chances of healing

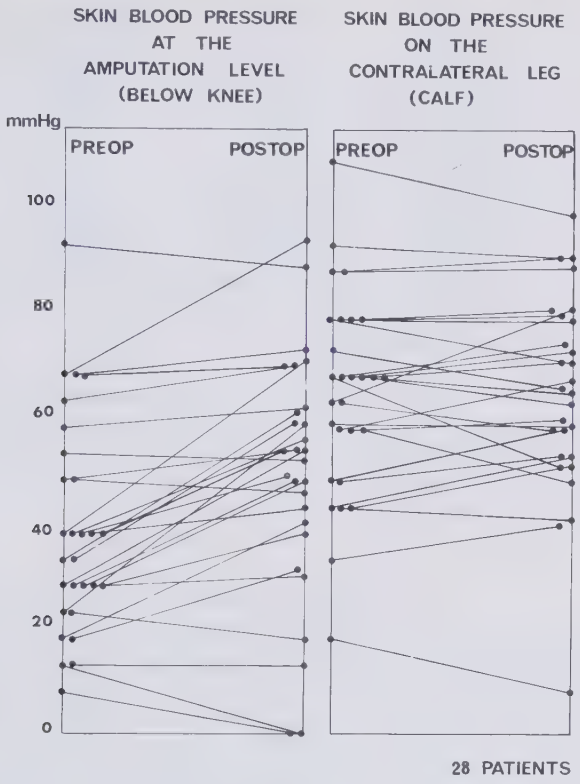

Figure 9. Skin blood pressure in 28 below-knee amputations.

Left column: Skin blood pressure measured preoperatively approximately 10 cm. proximal to the amputation level and again at the same site 2 to 8 weeks after surgery. The postoperative values are corrected parallel to changes in diastolic arm blood pressure (except from two zero-values). An increase in pressure after amputation is apparent in 11 cases ($p < 0.01$).

Right column: Pre- and postoperative skin blood pressure measured as a control on the contralateral leg usually on the calf. Postoperative values corrected parallel to changes in arm diastolic pressure. The values are fairly constant, S.D. 5 mm. Hg.

would be considered better on the basis of the postoperative values than on the basis of the preoperative value. This pressure rise is related to the surgical removal of a sizeable part of the distal runoff tissues. It occurs regularly if the amputation is made rather close to the site of the critical vascular occlusion.

One can preoperatively mimic this effect by applying a tourniquet at the level considered for amputation. With this maneuver ("pseudoamputation") the skin blood pressure proximal to the tourniquet will immediately rise approximately to its postoperative level. Unfortunately one cannot carry through this pseudoamputation while measuring with the radioisotopic technique. This is so because a long time (20 minutes or so) is necessary to measure the pressure, and the patients cannot tolerate this prolonged ischemia because of severe pains. For assessing skin pressure during pseudoamputation we routinely use a photoelectric technique (Fig. 10).[19]

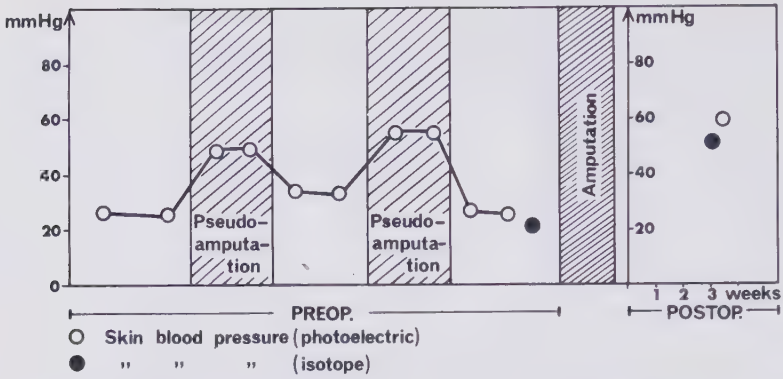

Figure 10. Skin blood pressure on the calf in a patient with gangrene of the foot measured with the isotope technique and a photoelectric technique. During pseudoamputation with a tourniquet, the skin blood pressure increased to about 50 mm. Hg. The procedure was repeated to secure reproducibility. Three weeks after successful below-knee amputation the skin blood pressure was again measured at the same site and the values corresponded to those obtained during the preoperative pseudoamputation (case 1).

CASE HISTORY 1

A 68 year old male suffered from severe resting pain and gangrene of the left foot. Normal pulses could be felt in the femoral arteries. The skin blood pressure measured with the isotope technique 10 cm. distal to the patella was 23 mm. Hg and about 25 mm. Hg as measured with a photoelectric technique. During pseudoamputation with a tourniquet at the level considered suitable for amputation, the skin blood pressure measured just proximal to the tourniquet increased to about 50 mm. Hg (see Fig. 10). A below-knee amputation was performed and healed primarily. Three weeks after surgery the skin blood pressure was again measured and values corresponding to those obtained during the preoperative pseudoamputation were found.

It should be noted that the standard technique of measurement with radioisotopes entails an element of pseudoamputation, because the cuff encircling the limb reduces the flow, but only a suprasystolic counter pressure will stop the circulation distal to the cuff completely and give a complete pseudoamputation effect.

Comments on the Routine Use of Skin Blood Pressure Measurements

The skin blood pressure is a measure of the *distal* arterial blood pressure. This distal pressure is, other factors being equal, such as the thermoneutrality and complete rest, a function of the *proximal* (aortic) pressure. We observe that the skin blood pressure in our patients varies in parallel with the variations in diastolic arm blood pressure. When this pressure is reasonably constant, then skin blood pressure is reproducible with a standard deviation of only 5 mm. Hg. This high accuracy is undoubtedly related to the integration caused by the prolonged observation periods of about 5 minutes at each of the "final" pressure steps made. If consistent variations in arm diastolic blood pressure are observed by repeated measurements, then a correction for such variations must be made.

The measurement of skin blood pressure has for some years been used routinely in our clinic for evaluating the prognosis and selecting the proper therapy. Spontaneous healing of small ischemic skin lesions is not uncommon. These patients are easily recognized by having a distal blood pressure of approximately 30 mm. Hg or more. They require no urgent therapy, but are usually candidates for elective reconstructive surgery.

We also see patients who suffer from intermittent claudication, and although they have no resting pain and no ulcerations, the distal blood pressure may be below 30 mm. Hg. This means that a skin lesion will not heal and often initiates a gangrene. These patients are therefore also potential candidates for reconstructive surgery. And, if other diseases or old age does not contraindicate surgery, an angiographic study should consequently be considered.

Surgery is of course the only radical way of repressurization of the distal arterial tree. But if reconstructive surgery cannot be performed or has failed, then a conservative medical treatment will in many cases result in healing of smaller chronic ischemic lesions in skin areas where the pressure is borderline with regard to wound healing, i.e., 25 to 35 mm. Hg. Simple classical measures such as elevating the head end of the bed and leg exercise as recommended by Buerger are helpful in improving the local circulation. Local skin compression must be avoided, and all types of drugs that tend to lower the systemic blood pressure and hence the distal pressure should be withdrawn if at all possible. In many old patients, stopping the use of diuretics may allow a small ischemic skin lesion to heal or rest pain to disappear. Edema should be prevented by avoiding dependent position of the legs for longer periods—not with diuretics. We consider sympathectomy to be contraindicated because it tends to lower an already critically low distal pressure.[18]

A more active form of medical treatment involves the elevation of the systemic blood pressure using the synthetic mineralocorticoid α-fluor-hydrocortisone (Florinef). By inducing moderate hypertension also the leg blood pressure rises and the ischemic skin lesions heal over some months if the local pressure can be maintained above 30 to 40 mm. Hg. In a recent series 51 out of 72 such treatments resulted in healing of chronic gangrenous ulcers which otherwise would have resulted in amputation.[14, 16]

In cases with the lowest skin blood pressure or where heart disease in preexisting arterial hypertension contraindicates the "medical repressurization," or in cases with already extensive necrotic lesions, amputation cannot be avoided. Here we employ the pressure measurements as a guide in selection of the proper level.[11]

CASE HISTORY 2

56 year old diabetic female with gangrene of the fifth toe on the right foot. Skin blood pressure on the dorsal side of the foot was 58 mm. Hg (arm blood pressure 140/70). A local resection of the necrotic tissues was carried out and the wound healed primarily (Fig. 11).

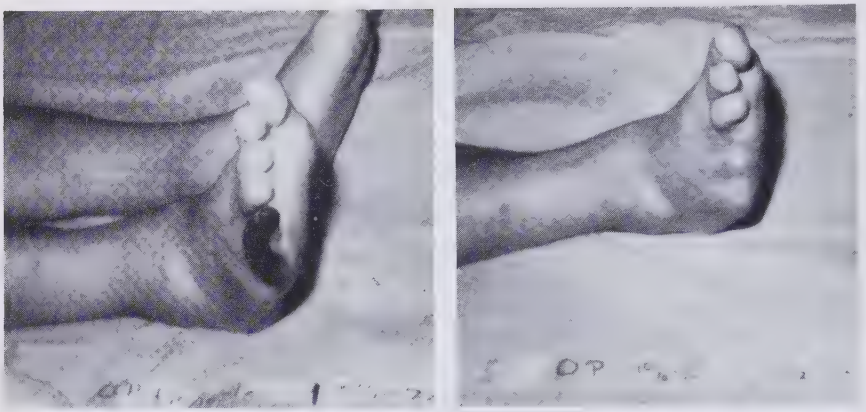

Figure 11. Pre- and postoperative appearance (case 2).

CASE HISTORY 3

51 year old male, above-knee amputee on the left side, now suffered from severe resting pain and gangrene of the right foot. Attempt of vascular reconstructive surgery was without success. The skin blood pressure on the calf was 13 mm. Hg and no increase could be registered during pseudoamputation. Despite this low pressure a below-knee amputation was performed in the hope of salvaging his remaining knee. Surgery was performed with meticulous care.

Postoperative skin blood pressure was unchanged at the low preoperative level. No sign of wound healing could be noted. There were no clinical signs of infection and repeated swabs did not reveal bacterial growth. Increasing skin necrosis and pain necessitated an above-knee reamputation 4 weeks later (Fig. 12).

To amputate as soon as the hopeless prognosis can be ascertained and at the correct level is mandatory for survival and the quality of the survival (general health and walking ability) of these often fragile patients.

Figure 12. Below-knee amputation stump 1, 2, and 4 weeks after surgery. Low pressure area with increasing skin necrosis and no wound healing (case 3).

This outline of the clinical use of skin blood pressure cannot do justice to many special problems. Diabetic skin lesions constitute such a problem. We just want to point out that skin lesions in diabetics heal just as in nondiabetics, i.e., if the skin blood pressure is above ca. 30 to 40 mm. Hg, provided infection is combatted and external pressure is not allowed to compromise skin circulation.

WHY USE RADIOACTIVE ISOTOPES?

The clinical diagnosis of occlusive arterial disease can be confirmed by a number of sensitive objective tests. In our opinion at least one such test should be used to confirm the clinical impression. The objective examination, in particular pulse palpation, is not sufficiently precise. The presence of palpable distal pulses does not rule out significant obstructive arterial lesions in the main artery. And as already mentioned, arteriography should not be necessary in most mild cases as surgical intervention is not indicated.

But how to choose the simplest and yet most reliable test? Shall we use oscillometry? Digital pulse morphology? Blood flow measurement with ^{133}Xe or plethysmography? Without giving a detailed discussion of such tests and their relative merits, we shall express the opinion that measurements of the ankle blood pressure by the use of the Doppler shift ultrasound detector[4, 24, 26] or, as in our practice, the mercury in Silastic strain gauge,[3, 8, 9, 22] which allows a measure of toe blood pressure too, are the best methods now available. These tests readily allow one to measure the whole span of pressures from over 200 mm. Hg to below 10 mm. Hg. Their diagnostic reliability is very high and, what is of great practical value, the results are expressed in millimeters of mercury, the clinical importance of which is immediately understandable for doctors and nurses, yes even for the patients.

However, we continue, in addition to distal systolic blood pressure measurements, to use the ^{133}Xe walking test in a great many cases. Objective assessment of the walking distance on a treadmill with adjustable speed and elevation is in our experience indispensable in claudicants. Then a ^{133}Xe flow study is easily added, and, in certain cases of branch thrombosis, only this test gives the correct diagnosis.

With regard to assessment of the prognosis in severe cases of leg ischemia, the distal *systolic* blood pressure measured by the strain gauge technique is also very useful. But in many such patients the pressure on the first toe cannot be measured because of ulcerations. The most important limitation, however, is in our experience the variability of the results in the lowest pressure range. If the cuff is applied too loosely the pressure is overestimated; if it is too tight the pressure is underestimated. Cases of increased wall stiffness also occur. Apparently in external compression of the microcirculation of the skin as in the radioisotope method, the pressure transmission from cuff interior (the small PVC cushion) to the venules of the skin is more reliable. Moreover, one cannot measure the blood pressure in a given skin area with the strain gauge; this value pertains to that large artery in the cross section encircled by the cuff which

has the highest blood pressure. It has not been our experience that we can predict the prognosis of ischemic skin lesions (and select amputation levels) with the same reliability with strain gauge as with the isotope method.

We are eager to abandon the skin blood pressure technique. To inject the isotope intradermally is in principle unattractive in gangrene and pregangrene cases. And the test is time consuming and causes discomfort. But then we must have another test that is safer and simpler, e.g., based on photoelectric recording of skin reddening as commented on above or another principle. To be without these pressures after having learned their value in patient management, we could not readily accept.

In this article we have emphasized the value of pressure over that of flow. Yet it cannot be disagreed that blood flow is what the tissues demand. Would it not after all be best to measure skin flow? No, probably not. Skin flow is very variable. What the blood pressure tells is the potentiality for flow: *when blood pressure is high, blood flow may also have the possibility of becoming high, i.e., of responding to increased tissue demand.*

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Nuclear Medicine Techniques in the Diagnosis of Deep Vein Thrombosis

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Improvements in diagnostic methods have shown that the incidence of pulmonary embolism is higher than previously documented. A recent review of 50 patients undergoing lung scanning in our laboratory revealed that 20 had clinical, laboratory, and scan findings diagnostic of pulmonary embolic disease.

Pulmonary embolism may be the first presentation of occult lower limb venous thrombosis. Autopsy studies have shown that 80 per cent of hospitalized patients have venous thrombosis and that one-half of the patients dying from trauma but without pulmonary embolism had venous thrombosis without antemortem signs.[20, 21]

Therefore, the diagnosis of venous thrombosis assumes an even greater urgency than previously appreciated. The diagnostic goals are to find the potential source of emboli or to locate their source once they have occurred.

Radionuclides have been used to study various stages of venous thrombosis. Tagged fibrinogen, given to patients at high risk for venous thrombosis, accumulates in a clot during its formation. Once formed, tagged white blood cells or fibrinolytic agents such as urokinase and streptokinase can be detected at the clot site. Radionuclide venography has been used to study venous dynamics in all phases from normal through occlusion and collateral channel formation.

Predisposing factors in venous thrombosis include stasis, infection, chemical irritation, trauma, hypercoagulable state, obesity, surgery, and pregnancy.[22]

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Table 1. *Radiopharmaceuticals Used in the Diagnosis of Deep Vein Thrombosis*

Measurement of tracer concentration
Fibrinogen tagged with ^{125}Iodine
Thrombolytic agents
Streptokinase-^{125}I
Urokinase-^{125}I
White cells tagged with ^{51}Cr
Imaging (dynamic or static) of tracer distribution
Radionuclide venography with simultaneous lung scanning
Macroaggregates-^{99m}Tc
Microspheres ^{99m}Tc
Radionuclide venography only
Pertechnetate ^{99m}Tc
Fibrinogen tagged with
^{131}Iodine
^{123}Iodine
Thrombolytic agents
Streptokinase-^{131}I, ^{99m}Tc, ^{111}In
Urokinase-^{131}T, ^{99m}Tc, ^{111}In
White cells tagged with ^{99m}Tc

METHODS

The radionuclide methodology applied to the study of venous thrombosis falls into two categories: (1) measurement of tracer concentration and (2) static or dynamic imaging of tracer distribution (Table 1).

Tracer Concentration

A detector is used to count the activity in the field of view. This is similar to the technique used for radioiodine thyroid uptake measurements. The application of these uptake techniques to the study of venous occlusive disease has included the use of tagged fibrinogen, streptokinase, urokinase, and white cells.

The uptake test provides information that may be displayed in graph or chart form. Many radionuclides may be employed for this test. However, if an image of the nuclide distribution is desired, then certain technical constraints exist. These include: (a) sufficient uptake in the area of interest to be able to distinguish it visually from surrounding structures (target-to-nontarget ratio); (b) the energy of the nuclide must be such that it is imaged efficiently by currently available detectors; (c) the emissions of the radionuclide must be sufficient to record an image in a reasonable time period without exceeding a reasonable radiation dose to the patient.

Fibrinogen

The identification of thrombi with labeled fibrinogen assumes that the injected fibrinogen will behave exactly as the unlabeled fibrinogen and be incorporated into a forming thrombus. It is then possible to locate

the thrombus by either external counting or imaging techniques.

The earliest published work using ^{131}I tagged fibrinogen for thrombus localization described the identification of an experimental thrombus in a laboratory rabbit.[6, 7] Modifications of this technique have gained wide clinical acceptance in Great Britain.[1, 2, 6, 13] As performed by Kakkar and associates, the patient is injected with 100 μCi of I–125 fibrinogen and using a handheld portable scintillation detector and ratemeter, the counts are obtained 2 in. apart along the calf and thigh and over the precordium at 1 and 24 hours. The data are plotted on a day to day basis. Leg to precordial ratios are established and followed daily.[9] The test must be run for several days as spurious results may be encountered if only the early counts are obtained.[2] A rise in counts over any area which persists for more than 24 hours is considered positive for thrombosis.

It has been reported that one third of patients develop venous thrombosis within 3 days after surgery and that of those, two-thirds had unilateral limb involvement.[5] The iodine–125 fibrinogen uptake test localized 83 per cent of the areas of venous thromboses found either clinically or by contrast venography.[9] In 50 per cent of the patients with documented thrombi, only the fibrinogen test was positive.

Negus and associates obtained a 93 per cent correlation between positive fibrinogen and positive phlebographic studies while negative studies correlated totally.[13] Unfortunately, tagged fibrinogen is not available in the United States for routine clinical use as the risk of hepatitis is considered prohibitive. Labeling of autologous fibrinogen is possible but a formidable task.

Fibrinogen studies utilizing iodine–131 require a more cumbersome detection apparatus because of heavy shielding requirements. However, earlier identification of thrombi is possible with this radiopharmaceutical. With the introduction of iodine–125 as the tag, the detector weight has been greatly reduced, permitting easy bedside examination. However, the low photon emissions of iodine–125 (27 to 33 KeV) precludes the imaging of deep structures with this nuclide and only uptake measurements are made.

Iodine–123, a cyclotron produced radionuclide, may prove ideal for use with fibrinogen because of its ideal energy for imaging (159 KeV) and its 13 hour half life. The radiation dose to the patient is reduced considerably. However, this nuclide has only recently become available.

Injuries to the lower extremities may produce false positive fibrinogen studies.[13] Therefore, the test is unreliable under these circumstances. Detection of venous thrombosis in the upper thigh and pelvis is extremely difficult because of high background radioactivity levels in the femoral artery and bladder. This limits the test to the diagnosis of calf vein or popliteal thrombosis.

In summary, radioiodinated fibrinogen is a useful agent for the diagnosis of venous thrombosis below the midthigh.[13] The use of iodinated fibrinogen is also useful in assessing the adequacy of thrombolytic therapy in deep vein thrombosis. This material may ultimately prove useful in the diagnosis of cerebral or coronary arterial occlusion, renal transplant rejection, and mitral valvular disease with suspected atrial clot formation.

Technical limitations at present make the routine use of this test impractical in most nuclear medicine laboratories. With the impending release of an approved fibrinogen preparation and increasing availability of short-lived radionuclides of iodine, this test may become more routine.

Thrombolytic Agents

In view of the problems cited above, work has gone forward attempting to locate formed thrombi using the labeled thrombolytic agents urokinase and streptokinase.

Urokinase has been shown to be an effective thrombolytic agent.[3] The great interest in urokinase has been due to its rapid blood clearance and lack of antigenicity. This material has been labeled with iodine-131 and technetium-99m and shown to localize in thrombi in dogs. It has not yet been used in humans.[16]

Streptokinase, labeled with iodine-125, iodine-131, technetium-99m, and indium-111, has been shown to enter thromboemboli as early as 15 minutes postinjection.[23] Dugan has been able to demonstrate streptokinase accumulation in emboli previously localized by other isotopic techniques.[5]

Both urokinase and streptokinase are difficult to label and require experienced personnel.

White Cells

Autologous white cells labeled with chromium-51 have been used to identify deep venous thrombi of the lower extremity. In 9 of 12 patients, the thrombus was localized accurately. Point counting along the leg and thigh similar to that of the fibrinogen uptake test was performed. The results demonstrated uptake at the site of thrombus as early as 1 hour postinjection. Maximum uptake was noted 12 to 24 hours postinjection.[12]

It was shown autoradiographically that tagged fibrinogen was layered on the surface of a thrombus while the tagged white cells were found throughout the entire thrombus. The labeled leukocytes clear from the blood more rapidly than the fibrinogen. This results in a higher target-to-nontarget ratio for leukocytes than for fibrinogen at the time of counting.[11]

The use of autologous white cells eliminates the risk of hepatitis that exists with commercial preparations of fibrinogen. However, the gamma ray energy (320 KeV) and low photon yield (8 per cent) of chromium-51 limit the image quality. The relatively high radiation dose to the patient limits the quantity of material that can be administered. One is therefore restricted to uptake measurements.

Recently, labeling of white cells with technetium-99m has been accomplished (M. A. Dugan, personal communication, March 1973). Hopefully, this will yield high quality images at low radiation exposures and ultimately lead to an additional useful diagnostic tool.

The agents discussed above for the localization of thrombi have the advantages of being natural components of the thrombus or natural antithrombus agents. Since thrombi can be imaged at a specific location, one could follow resolution or extension of thrombi (and embolization should it occur) once the thrombus is labeled.

STATIC AND DYNAMIC IMAGES

Radionuclide Venography

Radionuclide images may be of a static (brain, liver, and thyroid scans) or of a dynamic nature (as cerebral radioangiogram, aortogram, or venogram). Dynamic imaging requires a scintillation camera. The field of view of such instruments is approximately 10 to 12 in. The data may be stored on video tape and processed in a variety of ways immediately or at a later time to obtain estimates of regional function or uptakes. The scintillation camera, due to its ability to image dynamic events, has been used to study the vascular system in various states.[10, 14, 17, 24]

In 1969, Webber localized experimentally induced venous thrombi using technetium-99m macroaggregates of albumin (MAA).[27] He also compared MAA, human albumin microspheres (HAM), and macroaggregated fibrinogen (MAF), all labeled with technetium-99m, and $Sn(OH)_2$, as thrombus localizing agents. His findings indicated that certain particles were attached or attracted to thrombi. An important observation was that a zone of "stand-off" from the thrombus existed with some preparations presumably due to electrostatic forces.[26]

Additional studies by this group in which MAA was injected into a pedal vein and the lower extremities scanned showed a 70 per cent correlation with contrast venography in ten patients. Of the patients with

Figure 1. Radionuclide venogram (RNV): normal iliac veins and lower inferior vena cava.

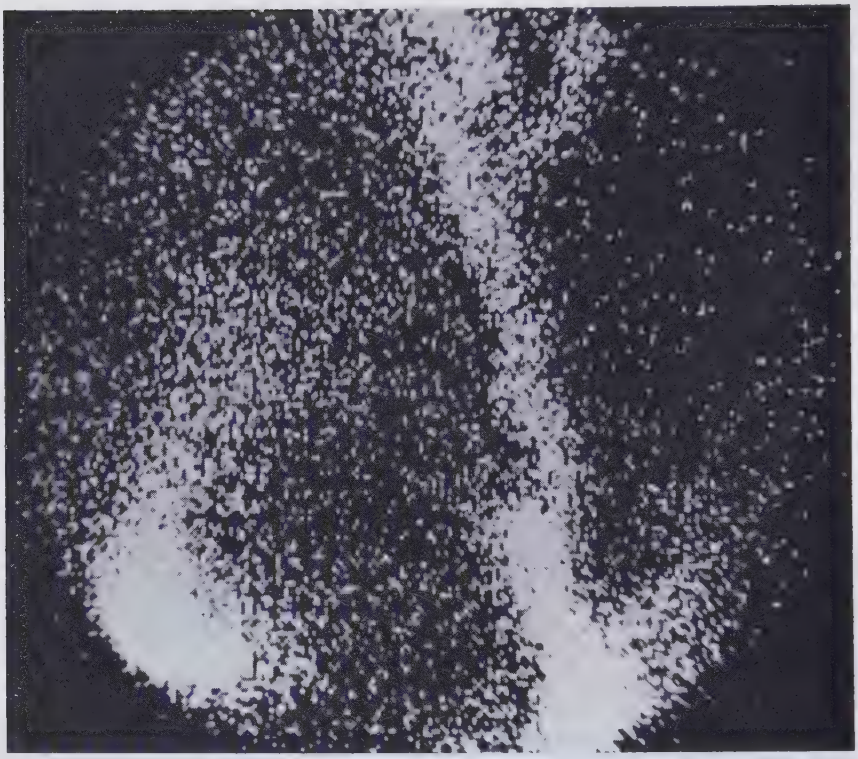

Figure 2. Obstructed inferior vena cava (same position as in Figure 1) showing collateral circulation in a patient with disseminated carcinomatosis.

lung scans positive for pulmonary emboli studied by this method, 80 per cent had positive leg scans as well.[25] However, there is an incidence of false positives by this method for reasons as yet not completely understood.

Rosenthall studied technetium–99m MAA for evaluation of the pelvic veins and inferior vena cava.[18, 19] Although only a small number of patients were reported, his work encouraged us to evaluate this method further.

Why do the MAA particles localize in the venous thrombi? It may be their irregular shape that causes them to be trapped mechanically or perhaps electrostatic forces cause them to be attracted to the vein wall or clot.[26]

The electrostatic charge theory is attractive since one can explain the "false positives" hot-spots for thrombus seen with MAA in the regions of venous endothelial damage. This damage leads to a change in the venous wall charge distribution which may attract all or part of the radiopharmaceutical to the injured region even though no thrombus is present. If this theory proves correct, the charge distribution or polarity of a radiopharmaceutical could be changed, altering its behavior relative to veins and thrombi.

MAA is irregularly shaped while HAM is spherical. The physical dif-

ferences between MAA and HAM may result in different charge distributions. Additionally, a detergent solution is used to suspend the HAM, which may also affect charge distribution (M. C. Crews, personal communication, February 1973). This may partially explain the decreased incidence of false positives when compared to the previous literature.

We have investigated technetium–99m HAM* as an agent for radionuclide venography. This material is a safe and excellent lung imaging agent.[4, 15] The conventional route of administration of this material is via an upper extremity vein. It is trapped in the lung capillaries and recirculation does not occur.

The radionuclide venogram is performed as follows: The patient is placed supine on a cart with a floating top. Two tourniquets are placed 1 to 2 in. apart just above the ankle of each foot and a butterfly needle (19 to 25 gauge) with a three way valve is inserted into a dorsal pedal vein. A scintillation camera fitted with either a standard 4000 hole or a 16,000 hole high sensitivity collimator and a videotape recorder is used for the study. The patient is positioned so that the following areas are in the field of view of the camera:

*Produced by 3M Company.

Figure 3. Pelvic collateral circulation in a patient with chronic venous disease post-irradiation (same position as in Figure 1).

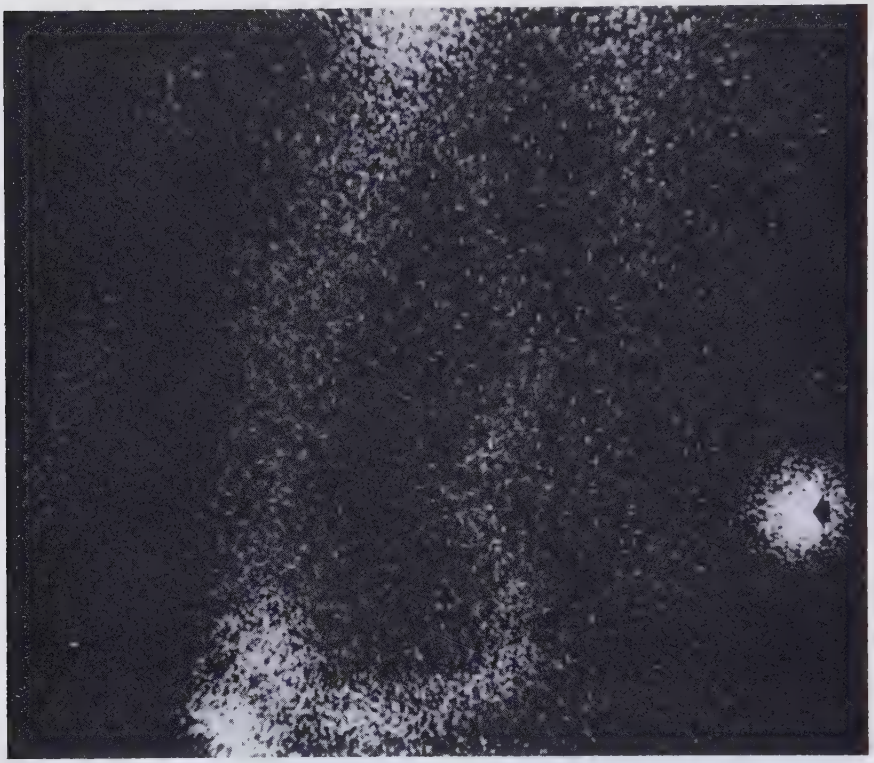

Figure 4. Collateral flow about the left popliteal vein and lower femoral vein in a patient with chronic venous disease. Black arrow indicates the location of a radioactive marker on the lateral patella edge.

(a) Iliac veins and inferior vena cava. An image of this region is made by injection of 1 to 2 mCi of ^{99m}Tc HAM bilaterally simultaneously followed by a 3 ml. flush of heparinized saline;

(b) Iliofemoral region. Injection of 0.75 to 1.0 mCi of ^{99m}Tc HAM unilaterally:

(c) Femoral vein. Injection of 0.5 to 0.75 mCi of ^{99m}Tc HAM unilaterally;

(d) Popliteal region (the lateral patella margin marked with a cobalt-57 marker). Injection of 0.5 to 0.75 mCi of ^{99m}Tc HAM unilaterally;

(e) Calf region (above the level of the tourniquets). Injection of 0.5 to 0.75 mCi of ^{99m}Tc HAM injected unilaterally. The same procedure (b through e) is repeated on the other side.

At the conclusion of the venogram, the needles are removed and a standard four view camera lung scan is performed.

All images made during the venogram are recorded on Polaroid or 70 mm. film and digital videotape simultaneously. Additional data in the form of transit times from injection until the first appearance of the radionuclide in the field of view are recorded.

The diagnostic categories established are:

Normal venogram (Figs. 1 and 5)
Suspicious venogram, increased transit time, flow abnormalities
Abnormal venogram
    Venous obstruction with visualized collaterals (Figs. 2–4)
    Venous obstruction without visualized collaterals
    "Hot spots" with or without obstruction (Fig. 6).

To date, 53 patients have been studied. Twenty-five patients had both x-ray contrast venography and radionuclide venograms with 96 per cent agreement between these two methods. A single disagreement occurred in a patient with an equivocal radionuclide study and venous occlusion on the contrast study. Injections were not made under pressure and only small volumes were used (maximum 1 ml. of radiopharmaceutical plus 3 ml. of saline flush). This may allow us to see the flow patterns which may be distorted when pressure injections are employed.

There was excellent visualization of collateral circulation. This difference from earlier reports is most likely due to technical advances in equipment since the earlier studies.

At present, our weakest diagnostic area is the calf. We feel that with further experience we may be able to evaluate this region more accurately. However, other techniques reported above may well be more accurate.

The ability to image pulmonary perfusion patterns provides either a baseline study in the patient with venous disease or an interval check on the progress of previous emboli. Of particular importance has been the virtual absence of false positive "hot spots." Until further investigation elucidates the exact mechanism, we are attributing this to the difference in electrostatic charge mentioned previously. It is in this area that the HAM is apparently superior to the MAA.

Figure 5. Normal femoral vein.

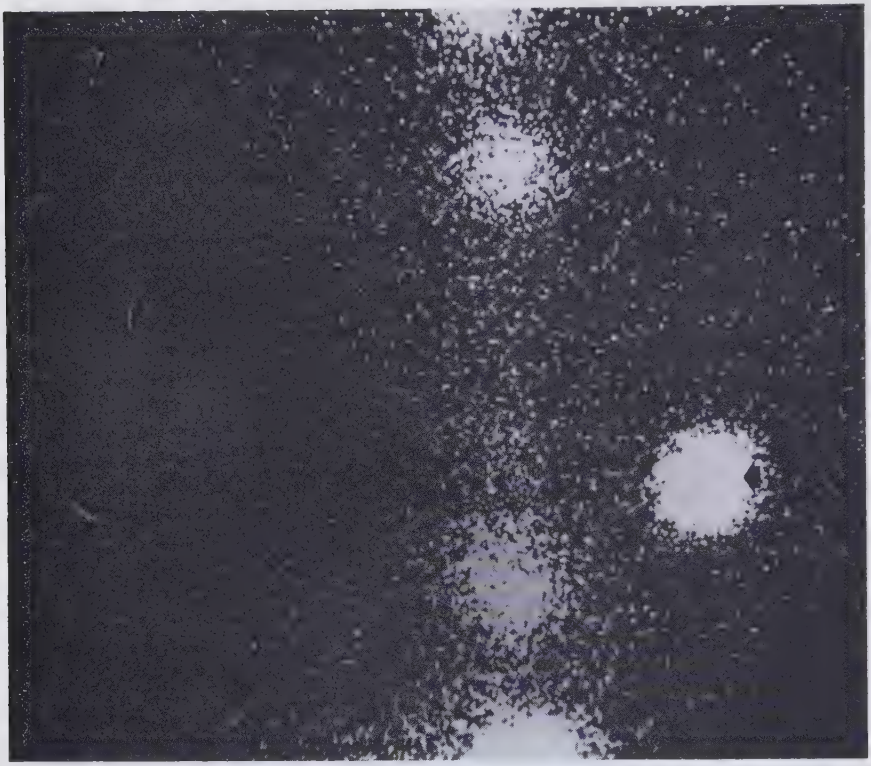

Figure 6. "Hot spots" suggesting thrombus formation in the left femoral and popliteal veins. Elderly female with pulmonary emboli without symptoms of venous disease. Black arrow indicates the location of a patella marker.

The advantages of this method over more conventional techniques include absence of hypersensitivity reactions to the injected material, a high degree of patient acceptance, the ease with which the technique can be repeated, and acquisition of information on the state of pulmonary perfusion. We have been particularly impressed with the utility of this examination in patients with pulmonary emboli without a known source (55 per cent positive venograms). The availability of scintillation cameras in many institutions should make this test readily available.

SUMMARY

In this "state of the art" review, we have presented the work of several groups. Many of the methods and materials discussed should be available in the near future. Other techniques are promising but require more laboratory work. By choosing the appropriate test or combination of tests, the clinician in conjunction with the nuclear medicine laboratory can best evaluate a patient with venous occlusive disease or pulmonary emboli (using radionuclide venography, white cells, streptokinase, or

urokinase), or follow high risk patients such as those going to surgery or those at bed rest for some time (iodinated fibrinogen or labeled white cells).

The use of these new methods in conjunction with more established techniques should result in identification of potential emboli and better assess the efficacy of antithrombotic therapy.

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Newer Techniques to Detect Venous Thrombosis with Special Reference to Electrical Impedance Plethysmography

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*and Alan B. Gazzaniga, M.D.***

Asymptomatic deep venous thrombosis of the leg continues to be one of the most common causes of death after surgery. Apart from the immediate risk to life, postphlebitic sequelae may occur consisting of swelling, varicose veins, ulceration, and trophic changes. There is evidence that early detection of deep venous thrombosis of the leg followed by anticoagulation can significantly reduce mortality and morbidity. Venography is a reliable method to detect deep venous thrombosis but is too complicated to employ as a routine pre- and postoperative screening procedure.

In recent years several new techniques for the early detection of deep venous thrombosis have been described and recommended as routine screening procedures. These include the use of the ultrasonic Doppler flowmeter,[13, 15] uptake of fibrinogen labeled with radioactive iodine,[6, 7, 11] and electrical impedance plethysmography.[10, 16]

We have found the Doppler method useful only in detecting complete or nearly complete deep venous obstruction and then only in veins above the knee. In addition, the Doppler technique requires a physician for implementation and thus is not suited to mass scanning. Details of the use of the Doppler technique are discussed elsewhere in this issue.

^{125}I–labeled fibrinogen uptake has been employed with considerable success in England for the detection of deep venous thrombosis but is not as well known in this country. The test depends on radioactive fibrinogen becoming incorporated in a forming clot and the increased radioactivity over the clot then being detected by an external counter. The patient's thyroid is blocked preoperatively by the administration of an iodide

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preparation. One hundred μc. ^{125}I-labeled human fibrinogen is then injected intravenously from 1 to 3 hours after operation. One hour later baseline counts are recorded over the thyroid, heart, and legs. These studies are repeated on days 1, 3, 5, 7, and 9. If a positive test is obtained, daily counts are made until the test becomes normal. ^{125}I-labeled fibrinogen is used rather than ^{131}I because its longer half-life (60 days as opposed to 8 days) facilitates storage and also ensures an adequate blood concentration for the period of postoperative monitoring.

The use of carefully screened single donor sources of fibrinogen has virtually eliminated the danger of hepatitis. Counting is made with a portable counter with the patient lying supine in his room bed with the leg elevated 10° above the horizontal in order to avoid venous pooling. No complications have occurred in a series of 65 patients receiving ^{125}I-labeled fibrinogen in our institution. The procedure has not resulted in any patient discomfort and has been employed in the seriously ill. It has been pointed out that the labeled fibrinogen test may be insensitive in detecting thrombi in the upper thigh and pelvis due to the overlying vascular muscle mass leading to false-negative results.[12] The presence of other concentrations of fibrin as that found in hematomas or healing wounds precludes the usefulness of this technique.[11] Thus, it is impossible to employ the fibrinogen uptake test to detect deep venous thrombosis in arterial leg operations or in patients undergoing hip operations.

All investigators are agreed upon the high accuracy of the fibrinogen technique to detect deep venous thrombosis confined to the calf, but its reliability in the thigh is not as readily agreed upon. Negus et al.[11] state that although most of their deep vein thromboses were confined to the calf, there was no difficulty in detecting independent thrombi arising in the femoral vein. Kakkar,[6, 7] Negus,[11] Frykhohn,[3] and Bauer[1] all feel that deep venous thrombi for the most part start in the calf and later propagate into the popliteal and femoral veins.

On the other hand Sevitt[12] states that calf-vein and thigh-vein thrombi usually arise separately and independently and that there are multiple independent sites of thrombus origin in the deep veins of the lower limbs and pelvis. Mavor and Galloway[8] are also of the latter school, namely that pulmonary emboli usually arise from thrombi which form de novo in the large veins of the proximal leg or pelvis.

ELECTRICAL IMPEDANCE

The use of electrical impedance to diagnose asymptomatic deep venous thrombosis was introduced clinically by Mullick et al.[10] in 1970. The technique is noninvasive, rapid and easy to perform at the bedside. The technique is based on the fact that blood is a good conductor of electricity. When a constant weak electrical current flows through part of the body, changes in the local blood volume alter the electrical impedance which in turn changes the voltage in accordance with Ohm's law (Voltage = Current × Resistance) (Fig. 1). In this test the patency of leg veins is assessed by observing changes in leg venous volume (manifested by impedance changes) during temporary venous occlusion induced by inspiration. The

OHM'S LAW

I = Current —— Flow (Rate)

E = Voltage —— Pressure (Gradient)

R = Resistance — Impedance

$$E = I \times R$$

Figure 1. Based on Ohm's Law, if current (I) is constant, then changes in voltage (E) are directly related to changes in resistance (R).

method introduced by Mullick and associates[10] requires the patient to breathe in slowly for about 5 seconds, after which he is told to hold his breath for 2 to 3 seconds and then exhale slowly. The test is repeated until two or three consistent readings are obtained. The single most useful measurement has been the maximum respiratory impedance excursion calculated from the highest point reached after inspiration to the lowest point occurring within 10 seconds after inspiration. Figure 2 compares a normal respiratory impedance excursion of 0.4 per cent on the unobstructed deep venous system of a patient's left leg with an excursion of only 0.11 per cent on the right leg which is the site of a deep venous thrombosis.

Mullick's technique, however, only evaluated one limb at a time. In normal patients the maximum respiratory impedance excursion almost invariably exceeds 0.2 per cent of the resting baseline impedance, whereas in patients with deep venous thrombosis the maximum excursion rarely reaches this value. Such unilateral limb evaluation is not only time-consuming but it lacks the advantage of being able to simultaneously compare both legs during the inspiratory maneuver. Gazzaniga[5]

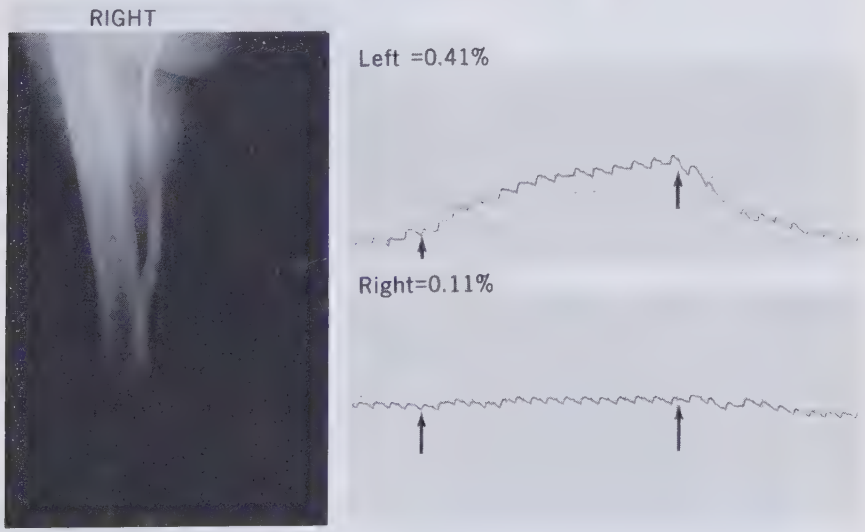

Figure 2. Comparison of right and left leg impedance excursions during inspiration in a patient with venous occlusion in one leg. Note that the impedance excursion on the left or unobstructed leg is 0.41 per cent while the right or obstructed leg has an impedance excursion of only 0.11 per cent. The venogram on the left confirms the venous obstruction in the right leg.

in 1972 introduced a rheograph for impedance plethysmography that measured impedance changes in both limbs simultaneously. The apparatus is called an impedance rheograph rather than an impedance plethysmograph since plethysmography by definition means volume changes. The rheograph does not measure changes in volume but changes in resistance which are related to volume changes. The rheograph* employed in our clinic has several advantages over that described and employed by Mullick and Wheeler (Fig. 3). Firstly, it is self-zeroing with internal mechanical calibration. Secondly, both of the patient's legs can be evaluated simultaneously, providing rapidity of testing and the use of the opposite leg as a control.

The excellent results of Mullick and Wheeler[10, 16] in detecting deep venous thrombosis by impedance rheography, however, have not been reproducible by other investigators[2, 4, 14] who have noted a significant incidence of false-positive responses employing this technique. The accuracy of the impedance results have been determined by venography. Our initial results with preoperative screening with impedance rheography showed an incidence of 8 per cent false-positive responses, but as more patients were studied the number approached 45 per cent. As a result of this high incidence of false-positive results, we investigated various res-

*Beckman Instruments, Fullerton, California.

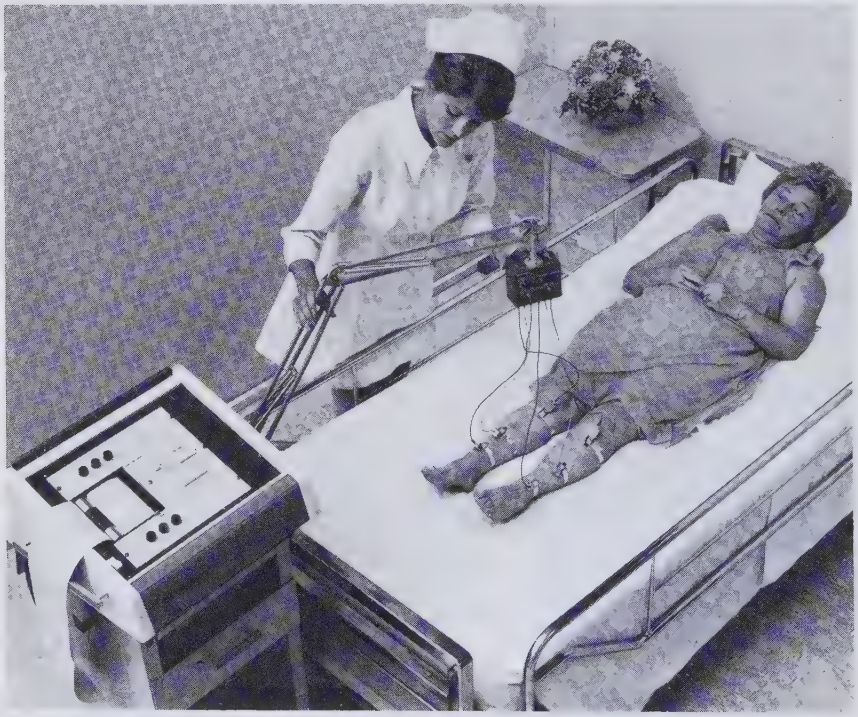

Figure 3. Impedance plethysmograph or rheograph employed in our clinic. It is self-zeroing with internal mechanical calibration and can evaluate both legs simultaneously.

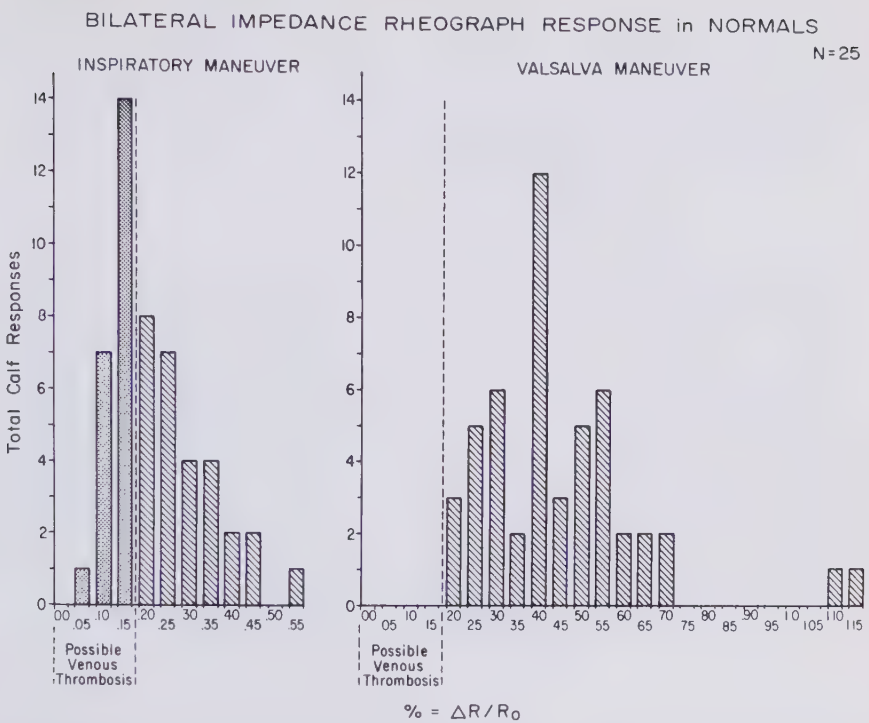

Figure 4. Comparison of impedance response in limbs of normal volunteers elicited by inspiratory and Valsalva maneuvers. The dotted boxes are deflections of 0.2 per cent or less using the inspiratory maneuver and by definition are abnormal deflections indicating deep venous obstruction. In contrast, on the right graph, no false-positive responses were elicited by the Valsalva maneuver.

piratory maneuvers and the Valsalva maneuver. Figure 4 compares the results of the two maneuvers in 25 normal subjects. Note the number of false positive responses with the inspiratory maneuver compared with the Valsalva maneuver. Twenty-two limbs of 25 volunteers had false positive responses with the inspiratory maneuver while none occurred with the Valsalva maneuver. Thus, there was an incidence of 44 per cent false-positives with the inspiration method.

Clinical Operative Screening

For clinical preoperative screening, impedance changes over the leg are first recorded after an inspiratory maneuver. If the impedance deflection is normal, the test is concluded. If the impedance deflection is abnormal, this result is confirmed by a Valsalva maneuver. The Valsalva maneuver appears to more consistently impede venous return than does simple respiratory inspiration. Figure 5 shows simultaneous femoral venous pressure and impedance recordings during inspiration and a Valsalva maneuver in the same patient. There is a more rapid rise in venous pressure to higher levels with the Valsalva maneuver than with inspiration. This more rapid and higher rise of venous pressure leads to greater venous pooling and a more marked change in basal impedance. The Val-

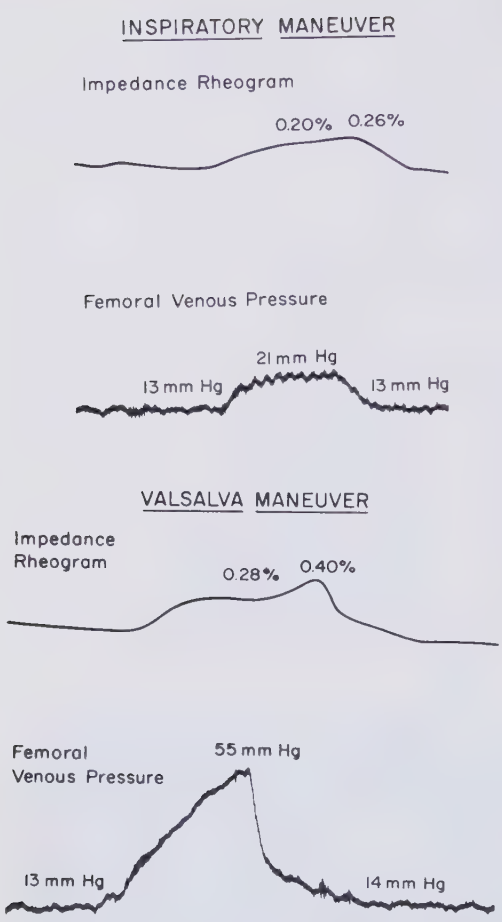

Figure 5. Simultaneous impedance excursions and changes in femoral venous pressure in the same patient elicited by an inspiratory maneuver and a Valsalva maneuver. Note that there is a more rapid rise in venous pressure to higher levels with the Valsalva maneuver than with inspiration.

salva maneuver is more consistent than inspiration in detecting venous obstruction since it uniformly increases right atrial pressure and impedes venous return.

False negative impedance results, on the other hand, are more difficult to resolve than are false positive results. If deep venous obstruction is on a chronic basis, superficial collateral veins can take over much of the venous return from the leg. Impedance plethysmography cannot distinguish differences in venous volume between the superficial and the deep venous systems. Temporary occlusion of the superficial system, therefore, should allow diagnosis of deep venous thrombosis by impedance maneuvers. Eight patients studied in our hospital with a history of recurrent or chronic deep venous thrombosis had normal impedance results with both the inspiratory and the Valsalva maneuver. These same patients had abnormal impedance responses when the superficial venous system was temporarily occluded by a tourniquet.[4]

This type of false negative response can be eliminated by the use of superficial vein occlusion, but the diagnosis of venous occlusion of the deep segmental veins below the popliteal veins provides the real current

challenge to diagnosis. Steer et al.[14] recently reported eight patients with false negative impedance responses who had thrombosis in these deep veins delineated by venography. We have been evaluating the ^{125}I-labeled fibrinogen test in a prospective manner before and after operation in patients and have found a much better correlation between the venographic presence of thrombosis with this test than with impedance studies.

Conclusions

It seems apparent that bilateral impedance rheography can be a useful screening maneuver for detecting major venous thrombosis of the leg. Acute or chronic venous occlusions can be diagnosed with the proper inspiratory or Valsalva maneuver when combined with the tourniquet control of superficial collateral vein channels. On the other hand, impedance rheography at the present time cannot detect thrombosis in the small deep veins of the calf. Diagnosis of this type of venous occlusion can be best made by the ^{125}I-labeled fibrinogen test. It may be that in the future a combination of the ^{125}I-labeled fibrinogen method and the impedance method may prove to be the best screening technique to detect deep venous thrombosis of the legs in surgical patients. Once the suspicion of deep venous thrombosis is made by clinical signs or one of the screening techniques herein described, venography is by far the most definitive method of elucidating the course of the disease.

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Intraoperative Monitoring of Femorotibial Bypass Grafts

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Bypass grafting to the tibial and peroneal arteries is an effective technique for revascularizing endstage ischemic limbs in those instances in which the popliteal artery is severely diseased.[3, 7, 10, 12] Prolongation of limb viability has been achieved in the majority of these patients, although the rate of graft occlusion is significantly higher than following aortofemoral or femoropopliteal arterial procedures. Numerous efforts to quantitate blood flow through arterial reconstructions have been carried out in recent years, in more proximal grafts.[2, 5, 8, 9, 11] However, there is a paucity of information regarding flow through femorotibial grafts.[4, 10]

Information derived from flow measurements may be of value for the following reasons. From a practical point of view, flow determined at the time of surgery may detect technical errors or inadequate inflow. Correction of these problems during the initial operative procedure can prevent graft failure and subsequent amputation. Data from these studies may also be helpful when correlated with the angiographic appearance of the outflow tract, to predict the longterm patency of grafts. Finally, flow measurement may be useful in determining whether there is a significant difference in flow dynamics between diabetic and nondiabetic extremities.

Bypass grafts to the tibial arteries have been carried out in 60 extremities. Intraoperative flow measurements were performed during 26 of these procedures in 21 patients, 17 of whom were male, and four female. Average age was 60 years. There were two grafts inserted in two patients, three grafts in one, and one patient was studied at the time of the initial grafting procedure and again at the time of the revision of a stenosis at the distal anastomosis.

The operative techniques employed have been described in detail previously and will be only briefly reviewed here.[3, 7, 12] Detailed angiography with delayed filming to demonstrate the degree of patency of the tibial vessels and runoff into the ankle and foot is essential before selecting patients for this procedure. Autogenous saphenous vein from the ipsilateral or contralateral extremity was used in the majority of these patients. Arm veins were used occasionally, when the saphenous veins

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were unavailable. A saphenous homograft was employed in one instance and bovine heterografts were inserted in four extremities due to the absence of suitable autogenous tissue. The tibial vessel selected for the distal anastomosis and the femoral artery were simultaneously exposed. A 1½ cm. arteriotomy was made and the vessel gently dilated up to 2½ or 3 mm. with graded arterial dilators. Dilute heparin was injected proximally and distally in the opened tibial vessel, and the distal anastomosis performed utilizing 6–0 monofilament polyethylene suture. A generous tunnel was created along the course of the patient's major vessels through the muscle compartment of the calf, the popliteal space, and into the subsartorial plane of the thigh. The graft was brought through this tunnel and the proximal anastomosis made to an arteriotomy in the common femoral artery. Occasionally, the proximal end of the graft was anastomosed to the mid superficial femoral artery, if the proximal portion of this vessel was widely patent. When the vein graft was not long enough to extend to the common femoral artery and the superficial femoral was partially or completely occluded, endarterectomy and vein patch angioplasty were performed on the upper portion of the superficial femoral artery down to the point of proximal anastomosis.

Technique of Blood Flow Measurements

Following the establishment of circulation through the graft, the patient's general hemodynamic status was carefully evaluated. Blood and fluid deficits were restored, and abnormalities of pH, Po_2 and Pco_2 corrected to insure adequate cardiac output and provide as normal a peripheral circulation as possible under the circumstances.

A previously placed radial artery cannula was connected to a Statham strain gauge transducer to monitor systemic arterial pressure during flowmeter studies. A Micron RC 1000 pulsed field electromagnetic flowmeter[13] was employed to measure flows through the vein graft. A noncannulating flow probe transducer of suitable diameter for a proper fit was applied to the vein graft. The flowmeter and probes were periodically calibrated in the animal laboratory, utilizing canine vessels and a volumetric control. The factor of error was less than ±10 per cent. Signals from the pressure transducer and flowmeter were fed to a 2 channel recorder for simultaneous permanent transcription of flow and pressure (Fig. 1).

Baseline flow through the graft into the leg and foot was recorded. Papaverine 10 mg. was then injected into the lumen of the graft to relieve peripheral arterial spasm incident to the operative trauma and prolonged exposure of the limb to the ambient temperature of the operating room. This technique has been suggested by several investigators to provide a truer picture of the flow capacity of the distal limb fed by the graft.[5, 8, 10] Optimum vasodilatory response to papaverine was achieved in 1 to 2 min. with a return to baseline flow after 4 or 5 min. following which the papaverine injection was repeated. Two baseline flows and two papaverine flows were recorded and averaged in each patient. Following this study, operative arteriography was routinely performed to demonstrate the distal anastomosis and runoff into the arterial bed of the calf and foot.

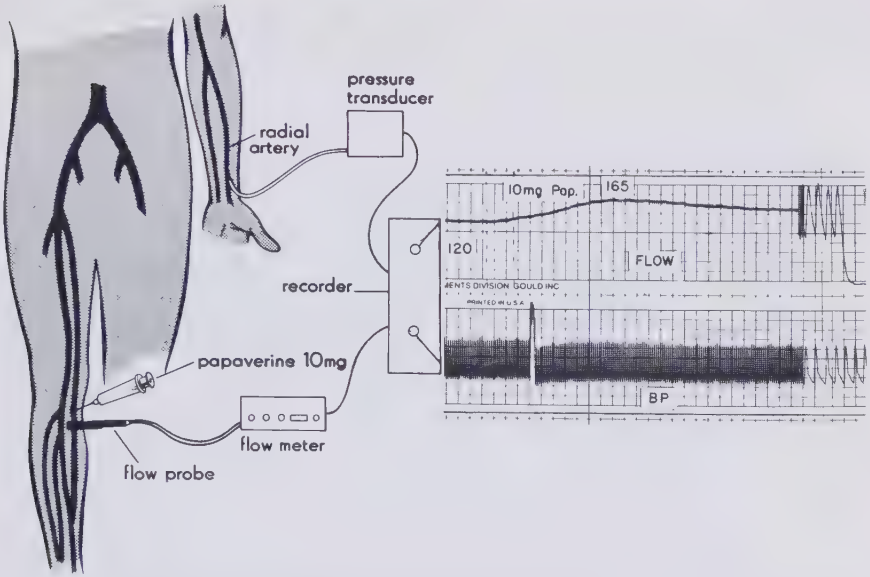

Figure 1. Simultaneous measurement of femorotibial graft flow and systemic arterial pressure is depicted in this diagram. After baseline flow has been recorded, papaverine 10 mg. is injected into the graft and peak flow is determined.

Results

The average baseline flow for all 26 studies was 90 ml. per min. (±36) with a range of 35 to 200 ml. per min. Average flow after injection of papaverine was 152 ml. per min. (±60) with a range of 35 to 300 ml. per min.

The results of these studies were compared with similar flow measurements performed on 25 patients undergoing femoropopliteal bypass grafts. Mean flows and one standard deviation for both graft types are portrayed in Figure 2. The average baseline flow for the femoropopliteal grafts was 100 ml. per min. and flow following papaverine flow volume increased to 184 ml. per min. It is remarkable that these flows were strikingly similar, although peak flow was somewhat higher in the femoropopliteal group. Using the *t* test, this difference was not statistically significant for baseline flow, and barely significant for papaverine flow ($p<.05$).

Blood Flow and Graft Patency

Postoperative follow-up of graft patency permitted grouping of grafts in the following categories: (A) graft thrombosis within 30 days, (B) early graft patency (over 30 days), (C) late graft failure (thrombosis between 30 days and 1 year), and (D) patency over 1 year. Mean intraoperative blood flows in each of these categories were compared and subjected to analysis by the *t* test for significant difference.

Graft thrombosis occurred in the immediate postoperative period in three patients (group A). The average baseline was 61 ml. per min. and papaverine flow 89 ml. per min. Twenty-three grafts were patent at the end of 1 month (group B) with an average baseline flow of 95 ml. per min.

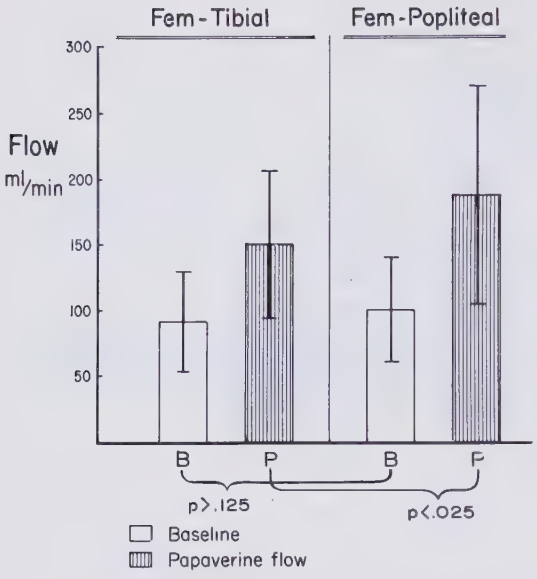

Figure 2. Comparison of baseline (B) and papaverine (P) flows through femorotibial and femoropopliteal grafts. The bar depicts mean flow and the vertical lines one standard deviation.

and an augmented papaverine flow of 158 ml. per min. The differences in baseline and peak papaverine flow between these groups were statistically significant. Nine grafts subsequently failed within the first year (group C). In this group, the average baseline flow was 92 ml. per min. and papaverine flow 147 ml. per min. There was no significant difference between the flows in these grafts and the early patency group (group B). Of those grafts which could be followed for more than a year (group D), nine remained patent with an average baseline flow of 100 ml. per min. and a papaverine flow of 173 ml. per min. Mean papaverine flow in this group was significantly greater than the flows in the early failure (A), early patency (B), and late failure (C) groups.

Diabetes and Graft Flow

The diabetic appears to have a somewhat different distribution of occlusive arteriosclerotic lesions, with more prominent involvement of the distal popliteal artery and upper tibial arteries. This probably accounts for the abnormally large number of diabetics among those patients requiring femorotibial bypass for relief of ischemia. The presence of degenerative changes in the microvasculature at the arteriole and capillary level has been presumed to be a major factor increasing peripheral resistance and reducing tissue perfusion. As a result, diabetics have been considered poorer candidates for revascularization procedures.

Thirteen of our patients, with 15 grafts, were diabetic and seven with 11 grafts were nondiabetic. Patients were classified as diabetic on the basis of an abnormal glucose tolerance test or abnormal 2 hour postprandial blood sugar. The baseline flow in the nondiabetics was 94 ml. per min. with a papaverine flow of 158 ml. per min., whereas in the diabetic baseline flow was 88 ml. per min. and papaverine flow was 144 ml. per

min. Though basal and peak flows were slightly lower in the diabetic than in the nondiabetic limbs, these differences were not statistically significant.

Discussion

The flow measurements reported in this study are strikingly similar to those presented by Kaminski and his co-workers.[10] In both of these studies, the volume of flow through femorotibial grafts was of the same order of magnitude as that achieved through femoropopliteal bypasses. These findings suggest that the capacity of the runoff vascular bed is similar for both types of grafts and is consistent with the clinical observation that both techniques are equally effective for the relief of ischemic symptoms. This lends further support to the rationale for the use of tibial bypass when the popliteal artery is obstructed.

The flowmeter has been demonstrated to be a useful tool for the identification of technical errors or inadequate inflow at the time of arterial grafting for occlusive disease. A flow significantly below that which might be expected, or a failure to respond to papaverine, strongly suggests mechanical obstruction.[4] Operative angiography is essential under these circumstances, as it will usually identify the problem and direct the surgeon to apply appropriate surgical techniques to relieve inflow obstruction, revise an unsatisfactory anastomosis, or remove em-

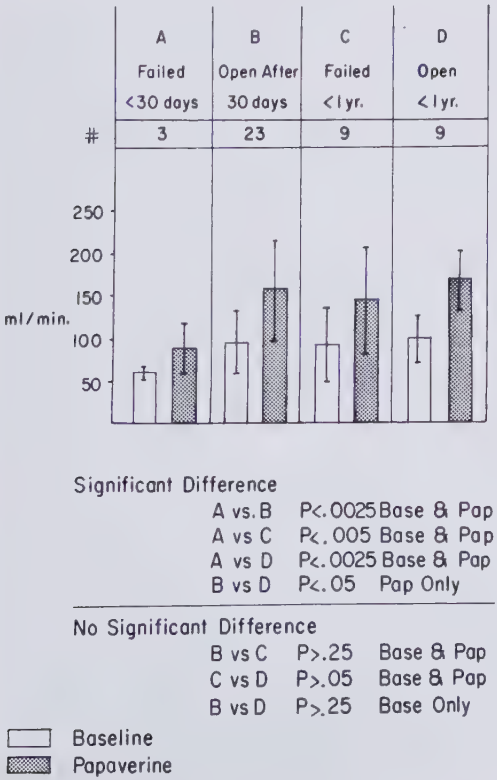

Figure 3. Comparison of baseline and papaverine flow through femorotibial grafts which thrombosed within 30 days (A), remained patent beyond the immediate postoperative period (B), remained patent initially but thrombosed within 1 year (C), and remained patent more than 1 year (D). The bar depicts mean flow and the vertical line one standard deviation.

boli due to operative manipulation. It is important to emphasize that palpation of a pulse, or even direct measurement of arterial pressure just below the distal anastomosis, may give erroneous information. We have frequently noted an excellent pulse proximal to a major obstruction.

The lack of correlation between flow and angiographic assessment of runoff has been alluded to by Mundth and his co-workers.[11] However, Kaminski and his associates[10] have shown a fairly good correlation between flow and angiographic demonstration of runoff. We have failed to affirm this correlation.[4] Further investigation with a larger number of cases will be required to settle this issue.

The ability to predict immediate and longterm outcome of a bypass graft by the measurement of intraoperative flow is likewise a controversial issue. Our results with tibial bypasses suggest that there is a modest difference, with higher flows correlating with longterm patency; however, the overlap between groups of occluded and patent grafts is so great that it is not possible to make an accurate judgment in the individual case. On this basis, it is probably unwarranted to consider the intraoperative flow measurement as an accurate prognostic test.

Graft flow in the diabetic does not appear to be significantly different from that in the nondiabetic, regardless of the difference in distribution of lesions in the cognate arteries or in the microvasculature. Our studies on both femorotibial and femoropopliteal grafts concur with those of Barner[1, 4] in this regard. The lesions frequently demonstrable in the microvasculature of the diabetic do not appear to restrict flow or reduce the response to vasodilators and, therefore, do not impair limb perfusion and cannot be directly implicated in the event of ultimate graft failure.

The use of papaverine to assess true flow capacity of an extremity at the time of surgery seems to be relatively well founded in the work of Hall.[8] Using implanted probes, he demonstrated that intraoperative papaverine flow correlated well with flow measured during exercise 1 to 2 days after surgery.

Although our studies suggest that low baseline and peak papaverine flows at surgery have a significant correlation with early thrombosis, the minimum flow required to maintain graft patency was not clearly defined. Some grafts with rather large flows at surgery became occluded in the early postoperative period, and other grafts with low flows have remained patent during this critical period. On the other hand, it is apparent that flows measured at surgery were sufficient to maintain patency in the majority of grafts and provide adequate perfusion to an ischemic extremity and support the healing of necrotic lesions.

Late graft occlusion in many instances is probably related to many factors in addition to surgical technique and the adequacy of the runoff bed. Frequently, the graft required to span the occluded segments of the femoral, popliteal, and tibial vessels requires the operator to use a saphenous vein of considerable length and of rather narrow diameter for some distance along its course. A rather significant correlation between graft patency and diameter has been established by Baddeley.[1] In addition, progressive arteriosclerotic changes in the inflow or outflow bed, especially in the diabetic, are common. Degenerative changes in the graft itself, secondary to operative trauma and anoxia of the graft wall due to

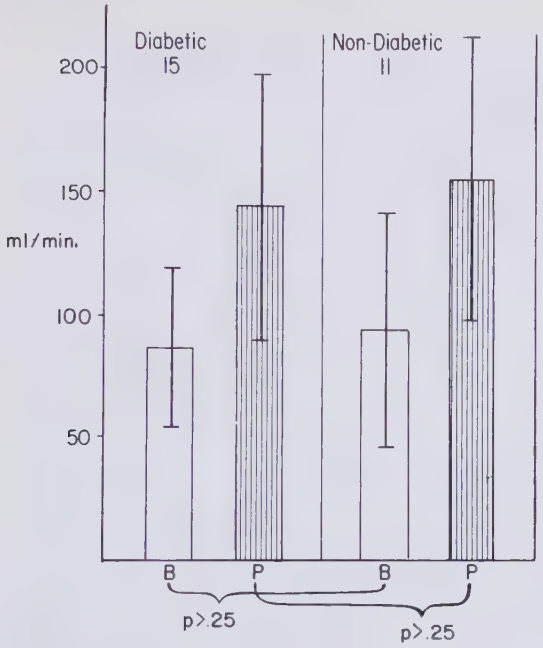

Figure 4. Comparison of baseline and papaverine flow through femorotibial grafts in diabetic and nondiabetic patients.

interrupted flow through vasa vasorum, may lead to fibrotic changes in the wall. Late obstruction due to fibrosis of a valve cusp or atherosclerotic degeneration of the graft have also been well documented.[6] Another significant factor is reduced cardiac output from concomitant heart disease. This may reduce flow below a critical level required for graft patency.

Finally, it is important to recall that the major value of many tibial grafts may be the provision of sufficient flow to heal necrotic lesions. If graft thrombosis occurs after this has been accomplished, the foot may have sufficient collateral supply to survive and be at least marginally functional. Though claudication returns, the patient is able to ambulate more effectively and with greater dignity than with a prosthesis. Under these circumstances, temporary patency of the femorotibial bypass graft has served its purpose, namely limb salvage.

Summary

The average flow through femorotibial grafts was 91 ml. per min. This increased to 152 ml. per min. following papaverine vasodilatation. These flow volumes are equivalent to those measured in femoropopliteal grafts.

There was no significant difference in flow between limbs of diabetic and nondiabetic patients.

Flow through grafts which became thrombosed early was significantly lower than the average and grafts which remained patent over 1 year had significantly higher flows than the average.

No correlation between flow and angiographic demonstration of runoff could be discerned.

The volume of flow measured at surgery is sufficient to relieve ischemic symptoms and maintain graft patency. However, minimum flow required to accomplish these ends has not been determined.

These studies support the use of tibial grafts for limb salvage when the popliteal artery is occluded.

The cause of late graft failure was not clearly defined by these studies; however, other factors in addition to the flow measured at the time of surgery should be considered.

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Quantitation of Flow in Femoropopliteal Grafts

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Reconstructive arterial surgery has now been in common clinical usage for almost two decades. Progress during most of this period has been dominated by development of surgical techniques and their long term assessment. By now, however, the operative approach has become reasonably standardised and greater attention is being paid to more critical assessment by quantitative methods at all stages in the management of patients with arterial obstruction. In the femoropopliteal segment the autogenous vein bypass graft is accepted as the most successful procedure but, nevertheless, the long term results leave room for considerable improvement.[8, 20, 27] To this end quantitation in terms of blood flow through the reconstruction may be important, and at the present time this is most easily achieved by intraoperative measurement with the electromagnetic flowmeter. The data obtained in this way may be of assistance in :(1) Providing evidence of either a satisfactory or faulty technical procedure. In the latter case immediate corrective action can be taken. (2) Providing a guide to long term prognosis based on the flow level achieved. (3) Giving indication as to whether accessory procedures such as lumbar sympathectomy should be added to the reconstructive procedure.

METHODOLOGY

The electromagnetic flowmeter is now established as a reliable means of blood flow measurement in blood vessels exposed at operation and such instruments have been used routinely in our peripheral vascular unit for the last 8 years.

Principle of Electromagnetic Flow Recording

To measure flow in an exposed vessel, a sterile probe is placed round the vessel and connected to electronic circuits which provide power for

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the magnet in the probe and process the voltage picked up by the electrodes in order to determine the flow.

The diagram in Figure 1 represents a cross-section through a probe on an artery. The electromagnet *M* provides a magnetic field across the vessel. A steady field would cause polarisation at the electrodes, so an alternating field is used. The magnet current and hence the magnetic field may vary sinusoidally (sine-wave excitation) or with a rectangular waveform ("square-wave excitation"), or may consist of pulses in each direction alternately, separated by intervals when the field is zero ("pulsed field excitation"). At present, instruments of all three types are in use. Their design has been discussed by Hognestad,[11] Westersten,[28] Goodman,[12] Wyatt,[29] and Gasking.[11] The frequency of the magnet excitation is usually between 150 and 2000 Hz. The flow of blood, or any conducting fluid, through the vessel causes a voltage proportional to the flow rate to be induced at right angles to the field and the direction of flow, i.e., across the diameter *a-b* in Figure 1. A slightly smaller voltage appears across the external diameter, where the electrodes *A*, *B* make contact with the vessel. The sensitivity of the flowmeter may be defined as the voltage induced between the electrodes for a certain rate of fluid flow. The effect of various factors on the sensitivity is discussed below.

Velocity Profile. Shercliffe[23] has shown mathematically that the flowmeter output is independent of the velocity profile, provided that it is symmetrical about the axis of the vessel. Thus it makes no difference to the flowmeter output, whether all the blood in a vessel is travelling at the same velocity – a "flat" profile – or whether it is moving faster in the axial stream than elsewhere, in a "parabolic" profile, for example. If the flow is

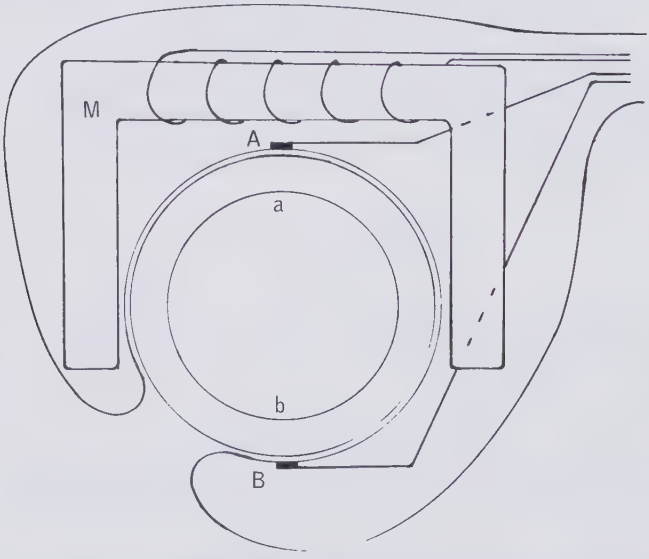

Figure 1. Cross section through a flowmeter probe on an artery. The electromagnet, *M*, generates a fairly uniform magnetic field across the artery. The flow of blood in the artery induces a voltage across the internal diameter *a-b*. The electrodes *A*, *B* in the probe make contact with the outside of the vessel. The voltage picked up is amplified and processed to determine the flow.

not symmetrical about the vessel axis, however, large errors can occur, as the flowmeter output is strongly influenced by the velocity of blood in the vicinity of the electrodes.[31] Jackson, Ellis, and Barnett[15] found errors of up to 23 per cent when measurements were made within 6 diameters of a 30° curve. In our own experiments, errors of over 50 per cent were observed when the probe was just downstream from an abrupt change in vessel diameter. When measuring flow at an operation it is, therefore, desirable to choose a straight, uniform vessel, well clear of an arterial junction or a sharp bend. Irregularities in the internal surface of a vessel, such as plaques, should be avoided if possible, or readings should be taken with the probe at several different sites in order to average the errors. A false reading may be too large or too small, depending on the relative positions of the probe and the obstruction to flow.

Despite the errors which may exist, measurements on irregular, diseased vessels can be valuable at the start of an arterial reconstruction, in order to confirm that a vessel is patent, or to check that there is no significant proximal arterial obstruction.

FIT OF PROBE ON VESSEL. If the probe does not fit the vessel snugly causing a slight constriction of the wall, large errors or a complete absence of signal will result. Spencer and Denison[24] recommended that the internal diameter of the probe should be up to 20 per cent less than the external diameter of the vessel for satisfactory measurement. We follow this practice, and measure the vessel before selecting a probe. This requirement means that a large number of probes of different diameters is required, so that the increment between one probe and the next is seldom larger than 20 per cent.

Probes are usually made with a narrow gap for insertion of the vessel, or with a wider gap which is closed by a shutter when the probe has been inserted. We prefer to use a probe with no shutter, but with a fairly wide gap, equal to half the lumen diameter of the probe, so that the vessel can easily be inserted without risk of damage. The slight bulge of the vessel at the gap is unlikely to cause an appreciable error.

ELECTRICAL CONDUCTIVITY OF BLOOD. There have been many conflicting reports on the effect of variations in electrical conductivity caused by, for instance, changes in the haematocrit of the blood. The theoretical aspects were set out by Wyatt.[30] They can be summarized as follows: The voltage generated by the flow appears across the internal diameter of the vessel, whereas the electrodes are in contact with the outside of the vessel. The voltage picked up by the flowmeter is therefore influenced by the relative electrical conductivity of the blood and the vessel wall. If one knew the thickness of the vessel wall and the conductivity of it and the blood, the precise correction factor could be calculated. Unfortunately, although conductivity of blood is easily measured or estimated from the haematocrit, the conductivity of vessel wall is variable, and depends on the amount of handling the vessel receives during surgery.[10]

We have calibrated probes in vivo on arterial vein grafts by connecting a cannula and syringe to a branch of the vein, and in vitro using freshly excised canine arteries or human veins and fresh heparinised blood.

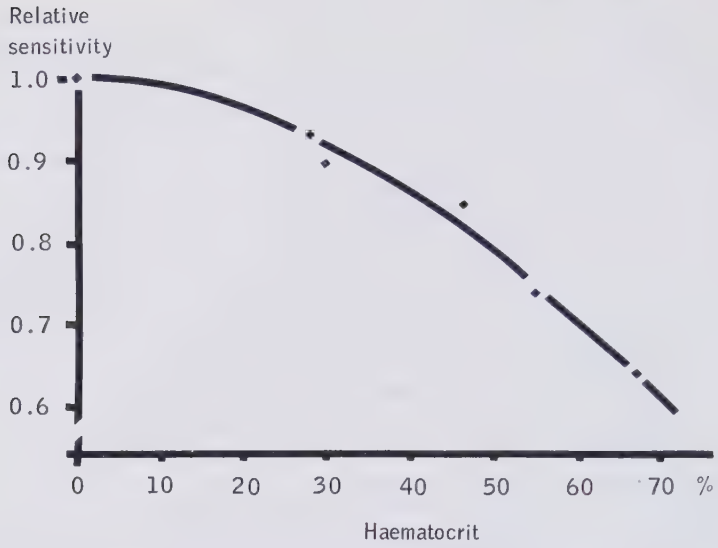

Figure 2. Variation of flowmeter sensitivity with haematocrit of the blood. The flowmeter was calibrated on freshly excised canine artery (which was found to give the same results as human vein) using fresh, heparinised, human blood. The haematocrit was varied by removing plasma or adding 0.9 per cent saline.

Our results from two typical probes are shown in Figure 2. These show that the sensitivity with blood of 55 per cent haematocrit is 26 per cent less than when normal saline is used as the perfusing fluid. This is in general agreement with measurements by Dedichen and Schenk[6] and other workers.

This and other evidence suggests that a blood vessel at operation may well have a conductivity similar to that of blood at about 25 per cent haematocrit. In that case, variations in thickness of vessel wall can be neglected at normal haematocrits, and the sensitivity when the haematocrit is 30 or 50 per cent will be within 10 per cent of the sensitivity at 40 per cent haematocrit.

Nature of Vessel Wall. At normal haematocrit, the thickness of the vessel wall has little effect on sensitivity, since the conductivities of vessel and fluid are similar. Spencer and Denison[24] demonstrated this by placing one artery inside another, thus doubling the thickness of the vessel wall, and showing that the sensitivity was unchanged.

Porous prosthetic arteries made of knitted Dacron appear to have a higher electrical resistance than normal arteries, as one would expect. In a series of calibrations using fresh human blood and Dacron grafts, we found that the sensitivity was increased by up to 16 per cent compared with the sensitivity on an artery or vein under similar conditions.

Measurement Procedure

The external diameter of the vessel at the site of measurement is measured with calipers. A probe having a lumen diameter of 0 to 20 per cent less than this is selected and fitted to the vessel. Some manufac-

turers recommend that the probe be first soaked in saline, but we have found this to be unnecessary with the probes we use (Nycotron Type PM). It is convenient to use a probe with a cable 2 metres long, so that there is no connector near the site of operation, where the contacts might be contaminated by blood or saline.

Before connecting the probe, all other earth connections from the patient including the surgical diathermy equipment are disconnected.[3, 13] This ensures that, even if a fault occurs, the patient does not receive a shock or electrical burn through the electrodes of the probe.

With the probe on the vessel and connected to the equipment, the vessel is closed for a few seconds, preferably several cm. distal to the probe, and the zero of the flowmeter is adjusted. A few seconds later, when the flow has returned to a steady level, the mean flow is read on a meter connected to the output of the flowmeter, and the waveform recorded by a pen recorder is examined.

At this stage, it is often useful to note the effect on flow of closing various distal branches in turn. For example, the popliteal artery can be closed first proximally and then distally to the distal anastomosis of a vein graft, in order to check the relative proportions of onward tibial flow and return flow via the proximal popliteal collaterals.

After use, the probe is rinsed, dried, and sterilized. Some manufacturers claim that their probes can be autoclaved, but there is no doubt that all probes last longer if autoclaving is avoided. We prefer gas sterilization using ethylene oxide. The initial cost of a range of probes of suitable diameters for most vascular operations usually exceeds the cost of the electronic equipment, but if autoclaving is avoided there are few probe failures in many years of regular use.

Detection of Technical Faults

Many surgeons have found that intraoperative flow measurements provide a rapid, convenient check on the adequacy of the arterial reconstruction,[3, 4, 18, 22] and we can confirm that a low mean flow or a "damped" waveform may indicate an unsuspected obstruction such as a plaque or thrombus in the popliteal artery, or a graft which is twisted or under too great a degree of tension. A low flow state may also be due to correctable hypovolaemia and we have noticed that marked flow variation with respiration is seen in this situation.[26]

Significance of the Flow Waveform

In general, the nature of the waveform in blood flow recording has received less attention than it deserves. Engell and Lauridsen[9] observed that momentary reversal of flow in the diastolic phase indicated significant physiologic vasoconstriction in the outflow tract. Figure 3 is an example of this phenomenon with brief flow reversal at the onset of diastole with almost zero flow in the remaining cycle. When this vasoconstriction is abolished by, say, lumbar sympathectomy the mean flow increases considerably,[25] and the dynamic trace reverts to the appearance shown in Figure 4, with forward flow occurring throughout the cardiac cycle.

A physical obstruction in a major artery acts as a low pass filter which attenuates the high frequency components of the flow waveform produc-

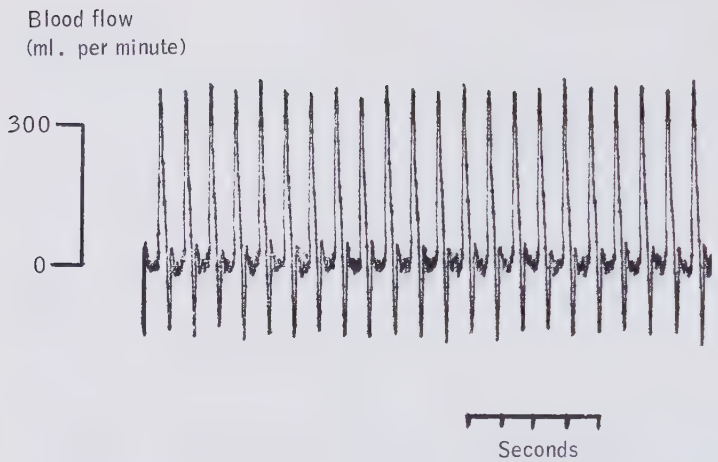

Figure 3. Blood flow in a vein graft in the presence of severe vasoconstriction. This gives rise to reversal of flow in the early postsystolic phase, followed by a very low flow. The mean flow was 47 ml. per minute.

ing a "damped" trace.[1, 7] This appearance is shown in Figure 5, where the amplitude of the systolic peaks is reduced and the rate of rise and fall is more gradual. The rate of rise and fall of the systolic wave component has been used to quantitate the degree of "damping" by Kouchoukos[16] and others.[4, 17]

"Damping" of the flow waveform is only apparent if the measurement is made below, or a short distance above, the site of the obstruction. Thus a measurement at the proximal end of a vein graft will indicate any inflow obstruction, but a stenosis at the distal end of the graft will give a waveform typical of vasoconstriction (see Fig. 3). It is therefore important to record from two sites adjacent to the proximal and distal anastomoses.

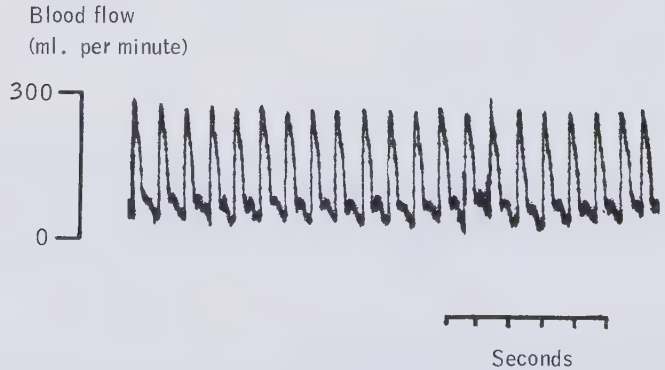

Figure 4. Blood flow in a vein graft supplying a vasodilated vascular bed. Flow is forward throughout the cycle, with a clear division between systolic and diastolic flow. Mean flow was 135 ml. per minute.

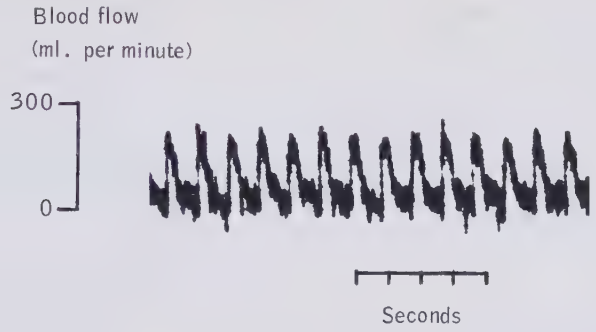

Figure 5. Blood flow in an obstructed vein graft. The effect of the obstruction is to reduce the peak flow, make the rise and fall less steep, and smooth out the flow waveform. Mean flow was 79 ml. per minute.

Detection of Arteriovenous Fistulae

It is easy to overlook small branches of an "in situ" vein graft, which act as arteriovenous fistulae if they are not ligated. Following the practice of Cronestrand, Ekestrom, and Hambraeus,[5] we check that the flowmeter indicates zero flow at the proximal end of the graft when the distal end is closed. If under these circumstances evidence of flow in the vein graft is obtained, the offending branch must be found and ligated.

In our hands measurement of flow provides the most convenient means of monitoring an arterial reconstruction. It adds only a few minutes to the length of the operation, and it will indicate any significant obstruction, whether proximal or distal to the site of reconstruction. Measurement of pressure alone in the graft can be misleading, as a normal pressure waveform can be recorded even if the outflow is completely blocked. The use of arteriography on completion of the reconstruction has been advocated for the detection of technical faults,[21] but we have found it to be more time-consuming and inconvenient than flow measurements, and there is the possibility of error if arteriography is limited to a single plane.

Prognostic Value of Flow Measurements

Reports on preoperative blood flow measurements in vein grafts have indicated a wide variation in mean flow with differing range of values from centre to centre. For example, Barner[2] reported values of between 17 and 130 ml. per minute, while Cappellen and Hall[3] considered the normal range to be 80 to 200 ml. per minute. These differences are perhaps to be expected when the multiplicity of variables which may affect flow is taken into account. Theoretically, providing the patient is normovolaemic and the cardiac output relatively unaffected by disease or anaesthetic agents, the "resting" mean flow in a technically acceptable vein graft is determined by (a) residual pathologic resistance in the tibial arteries and (b) the physiologic resistance of the microcirculation.

For comparisons of flow values to be meaningful, the effect of these factors must be minimized. The residual pathologic resistance is, of course, immutable, but methods are available by which the physiologic

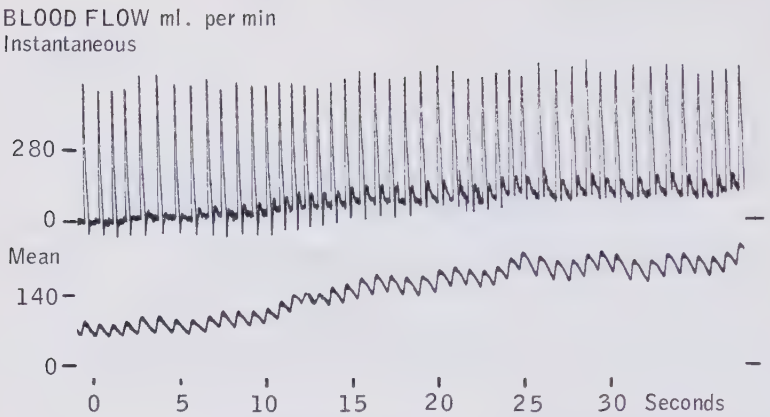

Figure 6. Instantaneous and mean flow in a vein graft following lumbar sympathectomy (at zero on the time axis). The flow waveform changed from vasoconstricted to vasodilated (Figs. 3 and 4) as the mean flow increased from 80 to 210 ml. per minute.

resistance can be lessened considerably and much higher flow readings obtained. Temporary vasodilatation may be induced by intra-arterial injection of such agents as papaverine[22] or reserpine.[9] A more permanent increase in mean flow can be obtained by adding lumbar sympathectomy[19] to the arterial reconstruction and we have found this procedure very effective in the majority of femoropopliteal bypass procedures[25] (vide infra). We have attempted to correlate peroperative blood flows obtained in this way with the later success or failure of the reconstruction.

A series of 58 femoropopliteal vein grafts was studied, the majority of which were done for severe ischaemia with impending gangrene of the foot. The results are shown in Table 1 and indicate a significant correlation between low flow rate at operation (< 100 ml. per min.) and failure within 1 year of operation. In this series the failure rate was 9 out of 15 (60 per cent) when the flow was less than 100 ml. per minute, and 9 out of 43 (21 per cent) with flows exceeding 100 ml. per minute (Mann-Whitney, U-Test, U = 189.5, p = 0.26 per cent). Despite this it was not possible to deduce a critical value below which graft failure would be certain.

Table 1. *Correlation of Peroperative Blood Flow Values in Femoropopliteal Vein Grafts and Patency at 1 Year*

	GRAFT PATENT AT 1 YEAR	GRAFT FAILED WITHIN 1 YEAR	TOTALS
Number of operations	40 (69%)	18 (31%)	58
Mean flow (ml. per min.)	199	122	
Range of flow (ml. per min.)	57–448	20–380	
No. of operations in which flow >100 ml. per min.	34 (79%)	9 (21%)	43
No. of operations in which flow <100 ml. per min.	6 (40%)	9 (60%)	15

Effect of Adding Lumbar Sympathectomy to Femoropopliteal Vein Grafts

In our experience,[25] the level of blood flow achieved once the major proximal pathologic resistance has been bridged by a vein graft, is significantly controlled by the physiologic component of vasoconstriction in the distal arteriolar bed. It is somewhat surprising that vasoconstriction can persist in a situation where previous ischaemia and recent cross-clamping of arterial inflow might be expected to produce a prolonged hyperaemia. In our patients the vasoconstriction could not be satisfactorily explained by an oligaemic state because adequate transfusion was routinely given. Of the many factors which may influence peripheral blood flow we suspect that general body cooling and, in particular, limb cooling may play an important role in the genesis of persistent vasoconstriction. Certainly whatever the initiating mechanism sympathectomy is usually successful in permanently abolishing the vasoconstricted state. In a minority of limbs sympathectomy is not followed by an increase in blood flow,[22] either because a significant degree of vasoconstriction was absent before denervation, or because fixed organic obstruction in the outflow tract was the major component of the distal resistance. When blood flow is measured with a probe placed on the vein graft during the performance of lumbar sympathectomy, a transient fall may be seen during isolation of the sympathetic chain followed by a sharp rise immediately after its division. This increase in blood flow is persistent and is accompanied by a change in the flow waveform from the vasoconstricted to the vasodilated type. This is illustrated in Figure 6, which shows the changes in the mean and dynamic flow in the first 40 seconds after division of the lumbar sympathetic chain.

SUMMARY

Peroperative measurement of blood flow by the electromagnetic flowmeter is now an established and reliable method of quantitation. When used with circumspection, the mean flow readings and flow waveforms obtained provide a valuable indication of the immediate haemodynamic situation and the probable late outcome of the vascular reconstruction.

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The Rationale for and Measurement after Profundaplasty

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The results of femoropopliteal reconstructions for atherosclerosis have been disappointing, and it would seem a 50 per cent patency overall after 3 years would be a fair assessment of results, though some have reported better results. Whether reconstructions have been effected by venous grafting or endarterectomy or a combination of each has, in our experience, made little difference to the early overall patency rate, though the long term results are better after autogenous vein grafting. Some have considered that failure of a vein graft is the reason, others that progress of a generalized disease is the cause. In a retrospective study Bouhoutsos and Martin[2] considered 300 consecutive patients who had been operated on by venous bypass graft in the years 1965 and 1966 and they related the results according to the age of the patients at the time of operation. Many of us have had the impression that operations below the age of 50 years were less successful over the long term than those over the age of 60 years, and this was found to be so. They also considered the number of patients in each group whose grafts were patent for 5 years or more (Fig. 1), and this indicated too that better results could be expected in the elderly rather than in the young, and so did the late amputation rate (Tables 1 and 2).

In another consecutive group of 610 nondiabetic male patients studied from 1961 to 1966, who had undergone reconstructive surgery of an aortoiliac and/or femoropopliteal segment, they found that over a 6 year follow-up, the amputation rate was significantly higher in the younger patients (Fig. 2) and that bilateral amputation was demanded in a far greater proportion too (Table 3).

If we consider the long term results of vein grafting of arteries after injury and after some conditions of arteries not associated with stenosis, it seems that autogenous vein is an excellent longlasting substitute for arteries. Murray[11] in 1945 demonstrated experimentally that autogenous

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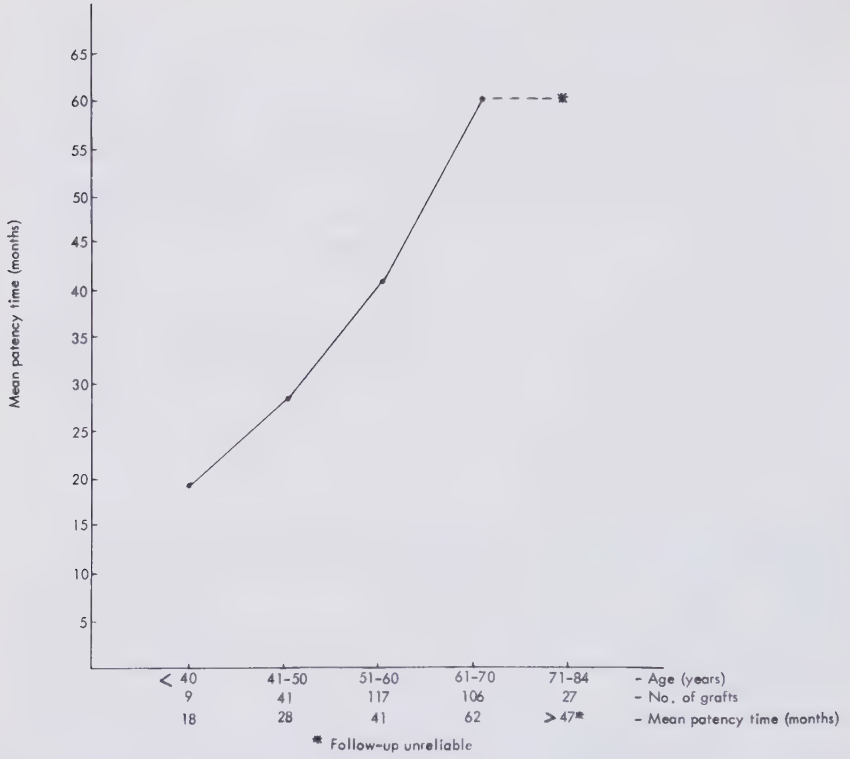

Figure 1. Success rate of 300 femoral popliteal vein grafts in relation to the age of patients (6 year follow-up).

vein is a suitable substitute for artery, and many have recorded apparently normal function of grafted vein after long periods for up to 18 years.[4,5] Similarly, after exclusion of a popliteal aneurysm not associated with stenotic disease, restoration of circulation by a vein graft has been successful and in 39 patients of ours so treated two remain patent after 12 years, with an average patency of 7 years and only one failure. In

Table 1. *Femoropopliteal Vein Grafts Patent for 5 Years or More*

AGE (YEARS)	TOTAL FEMOROPOPLITEAL VEIN GRAFTS	NUMBER OF GRAFTS PATENT AFTER 5 YEARS	PER CENT
33–40	9	0	0
41–50	41	3	7.3
51–60	117	18	15.4
61–70	106	29	27.4
71–84	27	Follow up unreliable	0
All	300 (273 + 27)	50	18.3

Table 2. *Longest Patency of Femoropopliteal Reconstruction in Different Age Groups of Patients*

AGE	LONGEST PATENCY TIME (MONTHS)
33–40	47
41–50	96
51–60	120*
61–70	120*
71–83	72*

*Still patent when last seen

addition, there has been no failure after vein replacement of a poststenotic subclavian aneurysm in 26 patients over the last 6 years.

We believe that if an autogenous vein graft for conditions not associated with progressive stenotic disease is patent and functioning approximately 1 month after insertion, it is highly probable that it will remain patent indefinitely.

Why then this difference between results of reconstructive surgery for atherosclerosis and conditions not associated with progressive stenotic disease? Surely it must be the progress of the disease and not the failure of the graft which is the important factor, and in this connection Royle (personal communication) has shown in the hyperlipaemic rabbit that atheromatous deposits are seen regularly at sites of injury, in this instance sites of anastomosis of an autogenous vein graft to the parent artery, and that obstruction at this site is common and early, which might give the impression that early failure of the graft was the

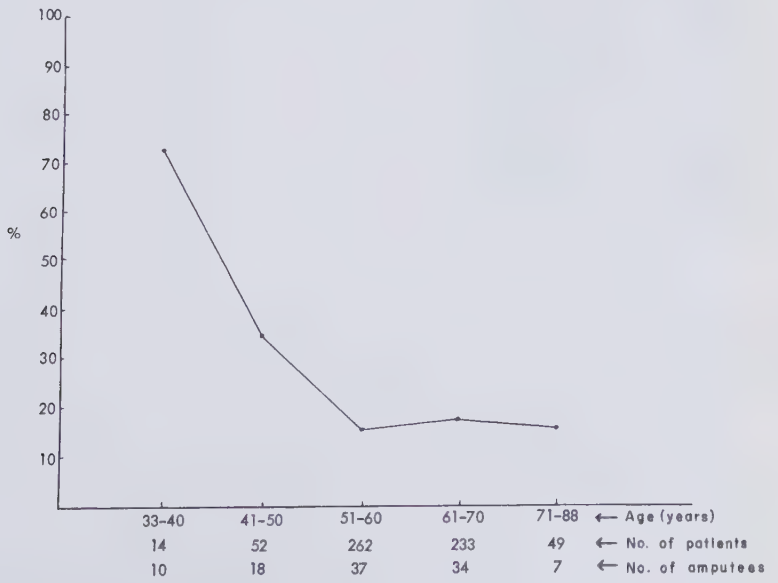

Figure 2. Rate of amputation in 610 arteriosclerotic patients in relation to their age (6 year follow-up).

Table 3. *Amputation Rate Among 610 Arteriosclerotic Patients (Males Nondiabetic) in Relation to their Age (6 Year Follow-up)*

AGE (YEARS)	NUMBER OF PATIENTS	NUMBER OF AMPUTEES		
		Unilateral	Bilateral	Total
33–40	14	4 (28.5%)	6 (42.8%)	10 (71.4%)
41–50	52	5 (9.6%)	13 (25.0%)	18 (34.6%)
51–60	262	28 (10.6%)	9 (3.4%)	37 (14.1%)
61–70	233	27 (11.6%)	7 (3.0%)	34 (14.6%)
71–88	49	4 (8.1%)	3 (6.1%)	7 (14.2%)
All	610	68 (11.1%)	38 (6.2%)	106 (17.3%)

cause of the obstruction. Because of this it might be that in the younger patient with abnormal lipoprotein metabolism a bypass graft is a disservice, as it may result in occlusion of collaterals.

In the younger patient it may well be that the disease is of a more florid nature, or it may be that as age advances, the disease process tends to die out. McManus[7] states that a previously atheromatous ulcer may be represented by a fibrous plaque in the 80 year old. Whatever the cause of the relative failure of femoropopliteal surgery, the fact is that this artery is the commonest one to be diseased, and indeed the one most often requiring surgery for the correction of ischaemic symptoms. In the younger age group, say those in the fifth decade, we avoid surgery of the femoropopliteal artery unless driven to this by incipient or early established gangrene.

Now the profunda femoris artery is a vessel which often escapes atherosclerosis except at its origin, and when it is affected it is later in life and to a lesser extent. Special angiography in the lateral plane, as has been shown by Beales et al.[1] at Hammersmith and Chelmsford Hospitals, has revealed the pathology of atherosclerosis as it affects this artery. They have shown that in all patients undergoing arteriography for ischaemic symptoms, 59 per cent have stenosis at the origin of this vessel from the common femoral, and that the stenosis may be confined to this site when it is often not visible on anteroposterior projection. In fact, in 27 of 46 limbs it was visible on lateral projection only (Fig. 3). Further they showed that disease may progress along this vessel to the first, second, or even the third perforating artery, but that distal to this the vessel appears radiologically normal (Fig. 4). In only 12 per cent of patients with lower limb ischaemia was the disease generalized throughout the course of the artery, and even in some of these the orificial stenosis was so tight that relief of this was clinically worthwhile.

Further, the anatomy of the profunda is such that there is an excellent anastomosis above with the cruciate anastomosis, and below with the recurrent tibial vessels. The arrangement of the branches is reminiscent of those of the superior mesenteric artery and the whole system is capable of dilatation if the superficial femoral artery is obstructed (Fig. 5). Figure 6 is the arteriogram of a London surgeon who had no symptoms in this leg, the profunda having enlarged, being free from disease

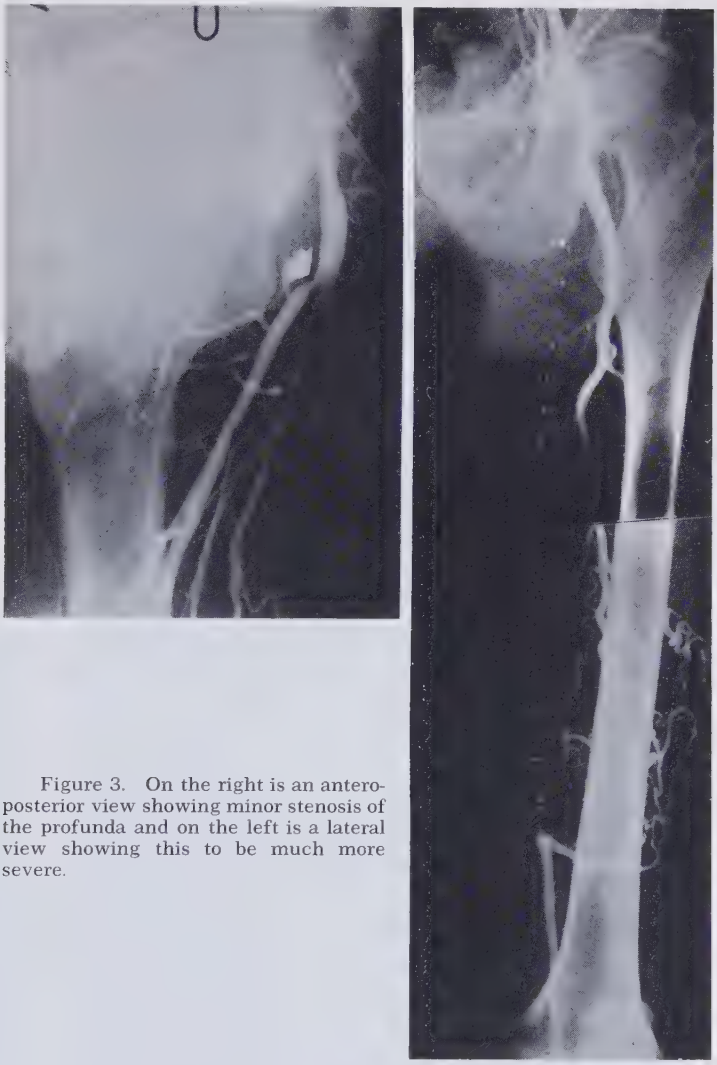

Figure 3. On the right is an antero-posterior view showing minor stenosis of the profunda and on the left is a lateral view showing this to be much more severe.

and able to carry sufficient blood for him to play 18 holes of golf without discomfort. This angiogram was part of an aortogram done for intermittent claudication in the other leg which showed iliac obstruction, after treatment of which he had no symptoms in either leg. It is anatomically an adequate substitute for the superficial femoral artery.

It is because of our better understanding of the pathology and of its anatomic suitability that we have concentrated especially on the operation of profundaplasty. It is a relatively minor operation which can be done under local anaesthetic if necessary. Others too have stressed the importance of this vessel.[8, 12, 14] We are very much averse to transfemoral unplugging of the orifice as has been advised by some because of the

Figure 4. An autopsy specimen showing gross disease of the common and superficial femoral arteries and of the first part of the profunda with healthy intima distal to this.

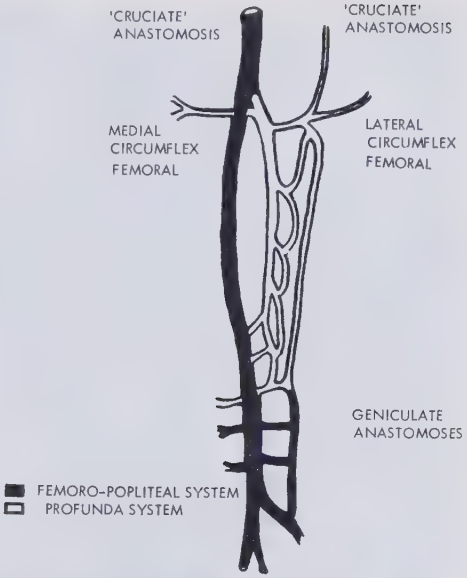

Figure 5. There are in fact four arcades.

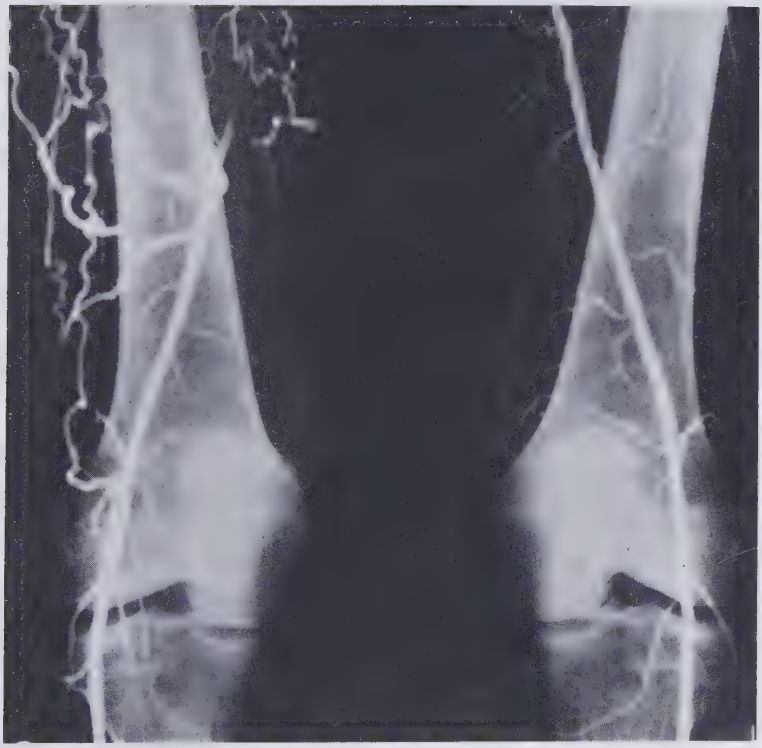

Figure 6. Angiogram done as part of an aortogram for intermittent claudication in the leg which showed iliac obstruction, after treatment of which the patient had no symptoms in either leg.

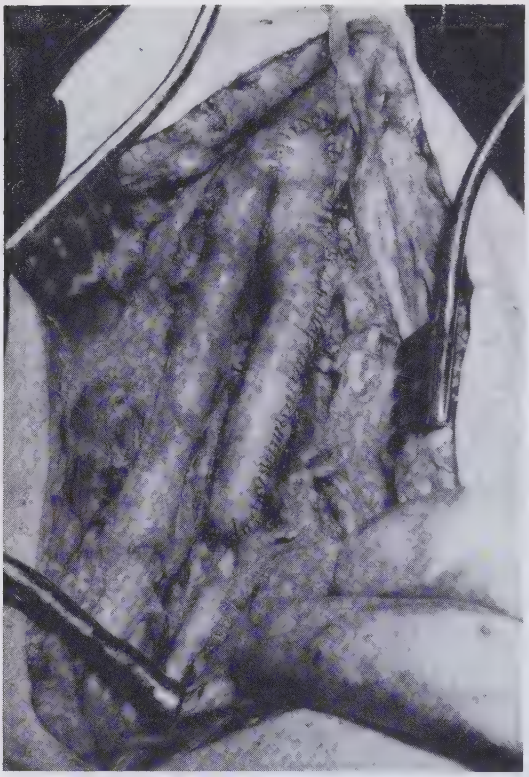

Figure 7. Exposure of the vessel freely by a wide arteriotomy and closure with a vein patch.

danger of incomplete clearance or because of a residual flap, and we are convinced of the necessity of exposing the vessel freely by a wide arteriotomy, and closure with a vein patch (Fig. 7).[10] It is for this reason that we have coined the term "profundaplasty."

Because of the poor long term results of reconstructions of the femoropopliteal trunk, especially in those in the fifth decade, we prefer to do a simple profundaplasty, and avoid bypass grafting, if a stenosis is shown on lateral angiography. We have found that in most cases, relief of claudication is such that the patient can walk say 500 metres or more, though his symptoms are not eased as dramatically as they are after a successful femoral reconstruction. But because of the relative freedom from atherosclerosis in the profunda, late failure is rare, certainly over a 3 year period. In the elderly with severe ischaemic symptoms, profundaplasty is more rewarding, though often requiring proximal iliac endarterectomy as well, and if this is done by an incision dividing the inguinal ligament and by a retroperitoneal approach,[13] the operation remains a relatively minor one with rewarding results. However, in the few patients over the age of 60 years who have no demonstrable profunda stenosis, a femoral artery reconstruction can be done with much greater hope of success over a long term than in the younger age group.

Clinical Results

In 1971 we were able to report on 217 limbs which had been operated on by profunda reconstruction and followed for 3 years. 173 had a proximal simultaneous reconstruction, most by endarterectomy, a few by vein or prosthetic ilio/aorto to common femoral/profunda grafts (Tables 4 and 5). 44 patients had profundaplasty alone, but the results in this group, though satisfactory, were not so striking (Table 6). The probable reason for the results in the latter group is that patients in this group had more distal disease than those in the former group to produce comparable symptoms. 61 of the combined groups were operated on for claudication alone, and 14 of these were not improved, and 3 of these who had a reconstructable lesion of the femoropopliteal trunk had this operation done later.

In substance therefore we use this operation in preference to femoropopliteal surgery for the patients with iliac stenosis, most of whom have a profunda stenosis too. In those below the age of 55 years with healthy proximal vessels and a good popliteal artery, we would do a profundaplasty if there is significant stenosis at the origin of the profunda. If there is not, then unless the symptoms are of threatening severity, we try to avoid surgery of the femoropopliteal trunk at this age though occasionally one is forced into this to preserve the life of the limb.

There is no doubt that a reconstruction of the proximal profunda remains patent over long periods, presumably because of the relative freedom from atherosclerosis of the main trunk of this artery. One patient 4 years after this operation obstructed his iliac artery. An endarterectomy of this was done and a subsequent operative angiogram revealed the original profundaplasty and the distal part of this artery more widely patent than immediately after the profunda reconstruction (Fig. 8).

Objective Measurement of Changes in Limb Perfusion after Profundaplasty

The changes in limb circulation which follow profundaplasty are not readily assessed clinically as distal pulses are not restored in 5 per cent of patients. The femoral pulse is unchanged. It is therefore necessary to rely upon the patient's symptoms in assessing the value of the operation and this index is not objectively recordable nor is it very reliable.

All patients in this series have been assessed by operative arteriography and radiographic improvement if a stenosis of the profunda femoris artery was obtained in all cases. The importance of operative angiography has been demonstrated by us on many occasions, as about 1 in 10 of all reconstructions of the vessel have been shown to be unsatisfactory and have required revision before wound closure. As lumbar sympathectomy was not used as an adjuvant to reconstruction, changes in flow can therefore be attributed to the reconstruction.

Six patients were investigated in a pilot study early in the history of this operation by measuring the flow in the artery after reconstruction by means of an electromagnetic flowmeter. They proved to have a mean flow rate of 213 ml. per min. which was significantly greater than the

Table 4. *Overall Results of Profundaplasty: 217 Limbs*

PROCEDURE	NO. OF LIMBS	NO. OF DEATHS	NO. OF AMPUTATIONS	NO CHANGE	SIGNIFICANT IMPROVEMENT	NO SYMPTOMS	EARLY FAILURES	LATE FAILURES
Profundaplasty with proximal reconstruction	173	0	16	8	100	49	19 (Amputations, 16)	0
Profundaplasty only	44	2	7	2	17	14	8 (Amputations, 7)	1

Table 5. *Profundaplasty Plus Proximal Reconstruction: 173 Limbs*

	BEFORE OPERATION	AFTER OPERATION	
		Results	Number
Distal gangrene	28	Healed, some with amputation of toe(s)	28
Rest pain	125	Relieved	110
		Amputation	15
Claudication	173	No change	8
		Significant improvement	100
		No claudication	49
		Amputation	1

Table 6. *Profundaplasty Alone: 44 Limbs*

	BEFORE OPERATION	AFTER OPERATION	
		Results	Number
Distal gangrene	8	Healed	5
		Amputation	2
		Death	1
Rest pain	31	Relieved	25
		Amputation	6
Intermittent claudication	44	No change	9
		Improvement	5
		Significant improvement	9
		No claudication	17
		Deaths	2

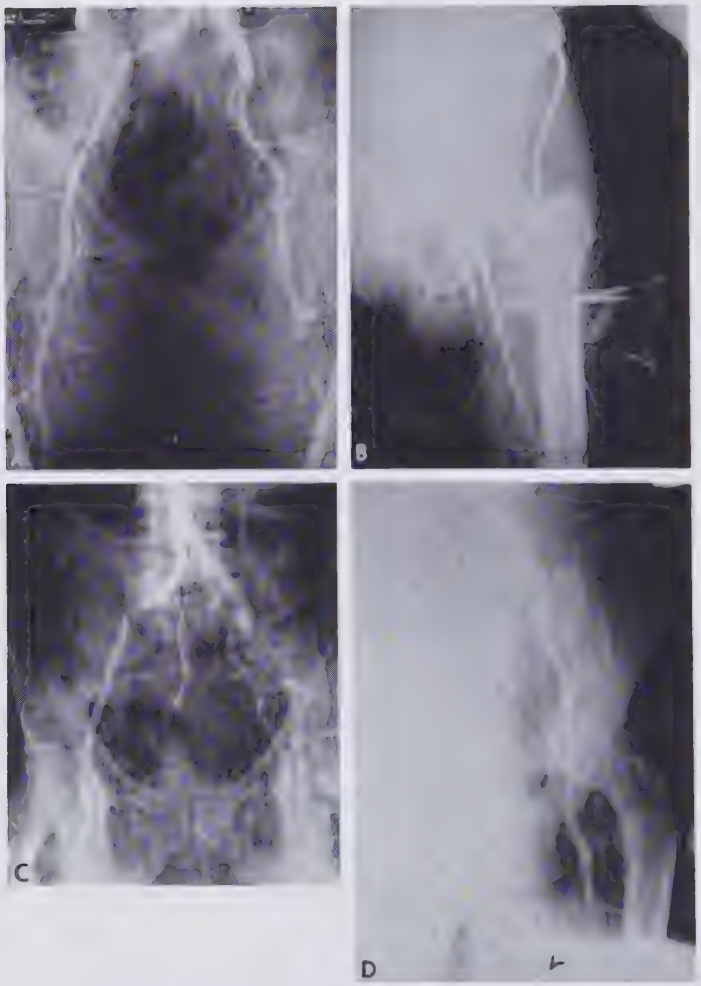

Figure 8. *A*, Original x-ray. *B*, Operative angiogram showing reconstruction of the profunda artery. *C*, Aortogram 4 years later when claudication recurred. *D*, Operative angiogram after clearing of the iliac artery without interference with the previously reconstructed profunda artery which has remained patent and satisfactory.

mean flow obtained in a reversed saphenous vein bypass of the superficial femoral artery.[9] The profunda, however, must supply both the thigh and the leg in these circumstances, but the femoropopliteal vein bypass supplies blood mainly to the leg alone. This is probably the reason for the high flow rates in the profunda in the presence of a superficial femoral arterial occlusion.

Cotton and his colleagues[3] have since published the results of studies in 46 patients in whom electromagnetic flow readings were obtained from the profunda femoris artery before and after reconstruction. They reported a 54 per cent increase in the mean flow from 106 ml. per min. to 163 ml. per min. and pointed out that a reading of profunda femoris flow during temporary occlusion of the superficial femoral artery may help to distinguish between the relative physiologic importance of a profunda stenosis and a coexistent superficial femoral arterial occlusion. Low flow rates indicate that the profunda stenosis is significant but flow rates approaching 160 ml. per min. indicate that the superficial femoral arterial occlusion is the more important.

These increases in measured flow through the vessel after reconstruction do not necessarily mean that flow to the calf muscles or the foot has been improved. At Hammersmith Hospital Bouhoutsos has investigated flow in the foot by volume plethysmography before and 2 weeks after the operation, and finally 6 months later. Data from 6 months after operation are not yet available but the 8 patients studied show highly significant increases in mean flow rates and occlusion pressure 2 weeks after operation.

Resting Flow		*Post-Exercise Flow*		*Occlusion Pressure (mm. Hg)*	
Pre-operative	Post-operative	Pre-operative	Post-operative	Pre-operative	Post-operative
2.5 ±0.6	9.9 ±0.8	7.5 ±2.1	15.6 ±7.6	68 ±13	108 ±23
$P < 0.001$		$P < 0.001$		$P < 0.001$	

Patients attending St. Mary's Hospital and operated upon by one of us (CWJ) have been assessed in the blood flow laboratory. The methods used have been reported.[6] The systolic pressure in the dorsalis pedis and posterior tibial arteries at the ankle was determined using an occlusive cuff and Doppler ultrasound probe placed on the skin over the artery. The systolic pressure can be expressed as an index of the brachial arterial pressure and it has been shown that low indices correlate well with severe occlusive disease. The patients were then asked to walk on a horizontal treadmill at 4 km. per hr. for 5 minutes or until stopped by claudication. The time at which pain occurred and the maximum time of walking was recorded. Ankle pressure studies were then repeated until the fall which accompanies hyperaemia in the ischaemic limb had recovered. The severity and duration of this fall in pressure were recorded. A large number of patients has been studied in this manner and the results have proved to be reproducible and correlate well with the severity of ischaemia as assessed clinically and by methods of measuring muscle blood flow such as xenon clearance.

The results of a series of 35 patients in which flow was restored to the profunda femoris artery by iliac arterial endarterectomy or bypass in patients with superficial femoral arterial occlusions has been published.[15] 27 showed objective evidence of improvement. Four patients with rest pain or distal gangrene were not improved and came to amputation and four claimed to have a decrease in their claudication but we could find no improvement in their walking distance on the treadmill or in the changes in ankle pressure accompanying this exercise. Xenon clearance studies were not performed. Thus this operation was accompanied by objective improvement in flow in 27 patients and subjective improvement in 31 of 35 patients. Very worthwhile results can therefore be obtained by restoring good flow to the profunda artery without treating a coexistent superficial femoral arterial occlusion.

The results of these studies in patients treated by profundaplasty alone have been more complex. 29 patients have been studied and 6 of this group had only moderate claudication and mean ankle pressures greater than half the brachial arterial pressure. They did, however, have a profunda stenosis observed on arteriography and confirmed at operation to be constricting the lumen of the vessel by at least 50 per cent. These 6 patients all claimed that their walking distance had improved after the operation but we could find no significant increase in their mean walking distance nor in the systolic pressure of the ankle vessels.

The remaining 23 patients were those who would be considered more suitable candidates for the operation. They all had severe claudication or rest pain or early gangrene and their mean ankle pressure was less than half their brachial arterial pressure. Six of these patients, all of whom had rest pain or gangrene, failed to show any objective improvement after profundaplasty. All thought their symptoms had improved for a variable short time after the operation but all came to amputation within 3 months. The other 17 patients showed a significant increase in their mean walking distance and resting ankle pressure when assessed 3 months after the operation and none has yet come to amputation. No significant further increase in these mean parameters occurred between 3 and 12 months after the operation.

Thus 17 of 23 patients with clinically severe ischaemia of the lower limb obtained worthwhile objective benefit from this operation but 6 lost their leg, in spite of the reconstruction of the profunda femoris artery, because of distal arterial obstruction. No patient presented with an objective increase in ischaemia after the operation of profundaplasty with or without iliac vessel reconstruction in this total of 64 patients studied to date.

Whether foot plethysmography or determination of the pressure recorded in the dorsalis pedis and posterior tibial arteries by a Doppler ultrasound probe is the more valuable measure of perfusion is not yet clear. Plethysmography measures total flow in the foot whereas ultrasound studies of the changes in pressure during exercise reflect more the flow to the limb above the ankle. Certainly plethysmographic findings are more in accordance with the subjective observations of the patients themselves. Which is more important, the pressure within the ankle vessels, or the ankle occlusion pressure is so far undetermined. Certainly the

ankle occlusion pressures seem more optimistic as to the results of the operation. However, the use of the treadmill often refutes in a practical way the claims of the patient that his claudication is less severe.

We are at present investigating patients undergoing profundaplasty by all the above methods, when it may well be possible to correlate the sometimes rather conflicting results of different measurements.

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Vascular Endoscopy

Possibilities and Limits of Its Clinical Application

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For half a century the endoscopic examination of blood vessels and heart chambers represented an attractive diagnostic and therapeutic challenge for many cardiovascular surgeons. Several remarkable efforts have been made to overcome the numerous technical problems with which Rhea and Walker were confronted when they attempted the first cardioscopy on dogs in 1913 (cited by Cutler, 1924).

In spite of many advances in the field of endoscopy such as replacement of blood by transparent balloons and perfusion under pressure (Brock, 1948; Sakakibara, 1958; Pinet, 1966), results were not satisfactory. Therefore, in the last decade cardiovascular endoscopy was nearly completely replaced by several other diagnostic procedures – angiocardiography, aortoarteriography, and venography.

A new impetus for further endoscopic studies was provided by progress in the field of reconstructive vascular surgery. The introduction of indirect methods for disobliteration of acutely or chronically occluded arteries and veins – ring or balloon technic; semiclosed thromboendarterectomy – gave rise to a need for direct visual inspection of the restored lumen.

Despite several clear advantages (shorter operating times, lower operative mortality, etc.), all semiclosed procedures, especially thromboendarterectomy, have one important disadvantage: the operation is done more or less without any visual control. Remaining clots or flaps of intima will often escape the usual intraoperative tests (see first three tests in Table 1). Incomplete disobliteration, especially of the femoropopliteal arteries or the iliac veins, represents the most frequent cause of early recurrence.

A new and improved technic of vascular endoscopy performed at the

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Table 1. *Intraoperative Tests for Vascular Patency*

"Flow test" by irrigation with saline solution
Passage of a ring stripper through the lumen
Inspection of the removed occluding cylinder
(Completeness? Change in the dissecting plane?)
Intraoperative angiography
Vascular endoscopy

end of the operation offers simple and safe lumen inspection. Our own technique is based on experimental and clinical investigations in progress since 1966.

GENERAL CONDITIONS FOR VASCULAR ENDOSCOPY

A good visual evaluation of the inner surface of blood vessels depends on the following conditions: (1) temporary interruption of blood flow; (2) replacement of the blood by a transparent medium; and (3) use of a sterilized endoscope of suitable length, diameter and good optical quality.

Instruments

There are two different types of instruments available: (a) A rigid angioscope with a Hopkins Optical System with a sixty degree angulated extravascular eyepiece; length: 41.5 cm.; external diameter: 6.3 mm., autoclaving possible.* (b) A flexible angioscope with a fiberoptic system. Length: 60 cm.; external diameter: 5 mm. Sterilization by ethylene oxide gas (Fig. 1).† Both instruments have an irrigation channel for the replacement of blood with a transparent saline solution and a connector for a suitable cold light supply.

Technique of Investigation

If both the distal and the proximal ends of the disobliterated vessel are exposed, the blood flow is interrupted by vessel clamping. In cases, however, in which only the distal (or proximal) segment is exposed we prefer intraluminal blocking, using a Fogarty balloon catheter (Figs. 2 and 6).

The endoscope is introduced through a longitudinal or transverse opening in the artery or vein. For the inspection of veins the instrument should be introduced in the direction of blood flow in order to avoid damage to the valves. For arterioscopy the instrument can be introduced in either direction. A retrograde control of the "run-in" through the most distal arteriotomy is generally preferred.

For every kind of vascular endoscopy complete asepsis must be guaranteed. A distance of at least 25 cm. between the wound and the eyepiece of the instrument should be maintained.

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†Olympus Optical Ltd., Tokyo.

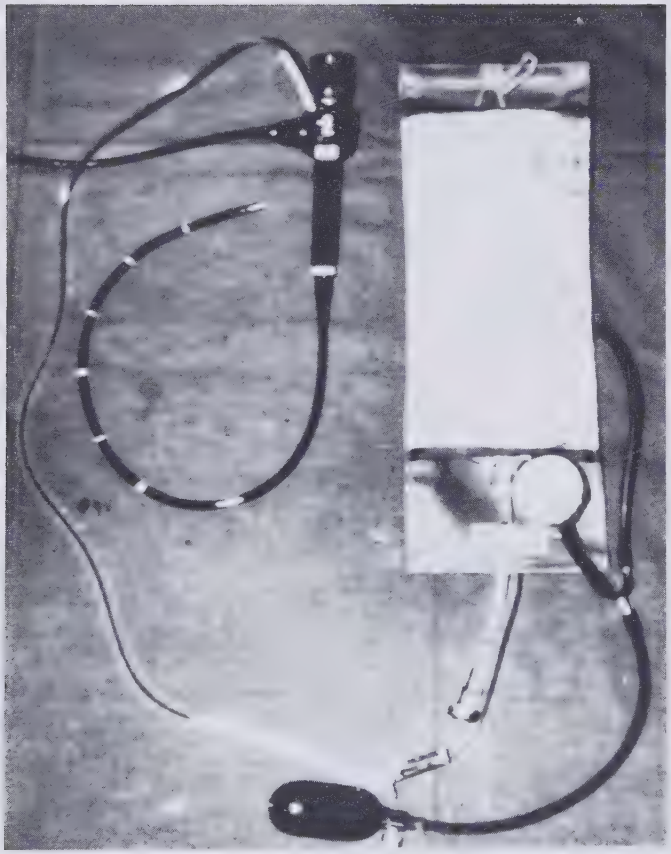

Figure 1. Flexible angioscope with a fiberoptic system (Olympus Optical Cholangioscope CHF). The instrument is connected with an Olympus cold light supply and a Fenwall pressure-infusion apparatus. Perfusion is done under controlled hypertension.

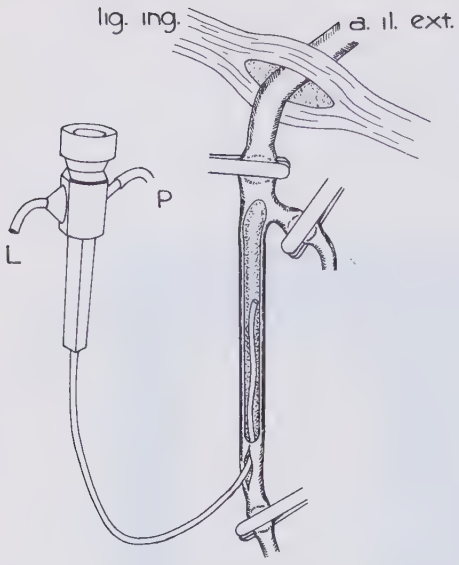

Figure 2. Technique of vascular endoscopy. In the femoropopliteal segment the bloodflow is interrupted by proximal and distal clamping. The endoscope is introduced from the distal incision up to the proximal clamping point. Inspection is done by withdrawing the instrument from the proximal to the distal segment under constant perfusion with saline solution. *L* = cold light supply; *P* = perfusion system.

Continuous irrigation of the vessel through the endoscope creates a transparent medium between the optical system and the vascular wall. The irrigation should be done under controlled hypertension in order to prevent collateral inflow (via uncontrolled branches), which would disturb the examination.

During endoscopy of the arterial system, the perfusion pressure should never exceed the systolic pressure. For venoscopy a pressure of 20 to 30 mm. Hg is usually sufficient to guarantee a satisfactory view. Uncontrolled hypertension can provoke distension and even rupture of the vessel wall. Especially in the endoscopy of a disobliterated artery in which the vessel wall consists usually only of the lamina elastica externa and the adventitia, a mechanical distension by high pressure perfusion can result in secondary rupture.

After careful introduction of the endoscope up to the proximal or distal blockage point, the lumen of the vessel can be inspected in whole length by drawing back the instrument to its insertion point. Sometimes it is useful to flush out the remaining blood by briefly opening the occluding clamps or balloons.

For documentation the endoscopic findings can be preserved by a movie camera or single pictures (Figs. 3, 5, and 7).

CLINICAL INVESTIGATIONS

The main indication for vascular endoscopy is to check the intraoperative lumen after disobliteration of occluded arteries or veins. Besides this surgical field vascular endoscopy will probably have no or only a small place as a diagnostic procedure in comparison to the several

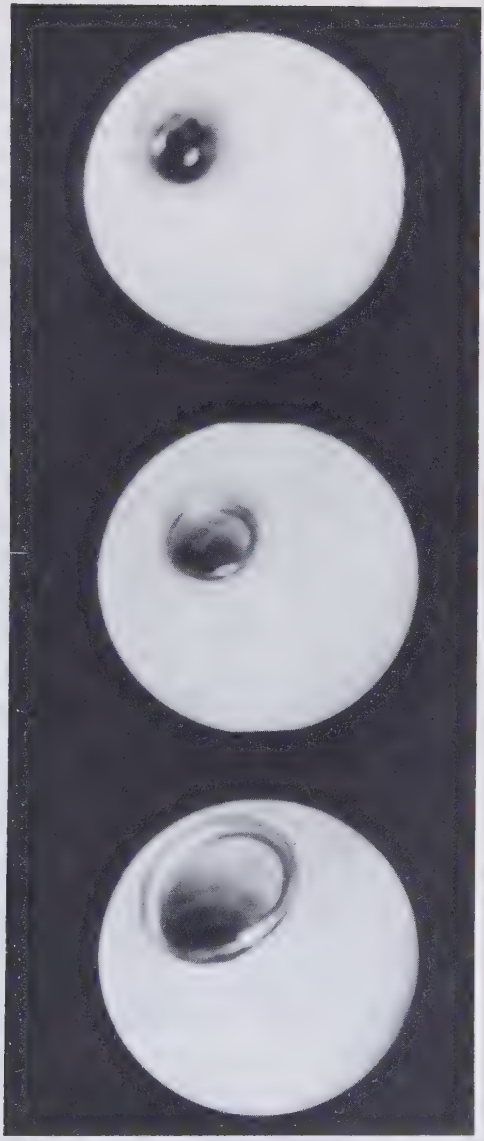

Figure 3. Lumen of a disobliterated superficial femoral artery. A ringstripper is introduced in the arterial segment which shows a smooth lining.

proved angiographic methods used for the morphologic assessment of the vascular status. Every type of vascular endoscopy needs surgical exposure of the main artery or vein. It is not to be expected that in the future a new development of vascular endoscopes which can be introduced transcutaneously will change that concept.

Endoscopy of Arteries

The most frequent indication is the intraoperative check of a semiclosed thromboendarterectomy of the aortoiliac and femoropopliteal arte-

ries. At the end of the procedure the disobliterated arterial segment should be inspected to exclude any possible technical fault (remaining intimal flaps, dissection of arterial wall, remaining clots). All these conditions can cause an immediate or early reocclusion of the vessels. If the disobliteration proves to be "incomplete" the lesion can be repaired immediately, either transluminally or by an additional arteriotomy.

Aortoiliac Segment. Generally the endoscope is introduced through an incision in the common femoral artery. Alterations of the aortoiliac segment such as kinking or elongation of the arteries make it necessary to use the flexible (fiberoptic) instrument. The inflow blockage is done by clamping of the aortic bifurcation or, if this segment is not exposed, by transfemoral introduction of the Fogarty catheter.

Femoropopliteal Segment. The endoscope is introduced from a distal incision in the popliteal artery up to the proximal segment of the superficial femoral artery. In nearly every case interruption of inflow is done by clamping of the femoral bifurcation.

Technical difficulties can arise if the caliber of the artery is less than 6 mm. (the endoscope cannot be introduced without the danger of mechanical damage to the artery). In those cases the result of disobliteration should be checked by intraoperative arteriography.

The perfusion volume during endoscopic examination in the aortoiliac and femoropopliteal area has been limited to 500 to 1000 ml. of saline solution. No deleterious effect of overloading the cardiovascular system was observed.

Endoscopy of the Venous System

It is used predominantly during surgical intervention for acute phlebothrombosis (venous thrombectomy). The endoscopic examination gives the surgeon clear information about the completeness of the disobliteration. The main application of venoscopy is in transfemoral thrombectomy for iliofemoral venous thrombosis. Besides surgery there is only a small place for venoscopy; for instance, in situations in which the conventional venogram fails to give a clear diagnosis. These can be an iliac "vein spur" or a post-thrombotic vein damage. The instrument is introduced from the groin through the common femoral vein or the saphenous vein, which can be ligated after the examination (Fig. 4). To prevent any collateral inflow, which might disturb the view, the common femoral vein has to be isolated completely. The "run-in" from the deep and superficial femoral vein can easily be interrupted by clamping. A Fogarty catheter is pushed up to the confluens of the common iliac vein to stop the central "run-off."

Venous endoscopy generally needs a higher perfusion rate than arterioscopy (1000 to 2000 ml. of saline solution). Also in the venous system the infusion should be done under continuous pressure control. The relatively high perfusion volume creates the danger of overloading of the cardiovascular system. Therefore good central blockage of the vena cava is a very important aspect.

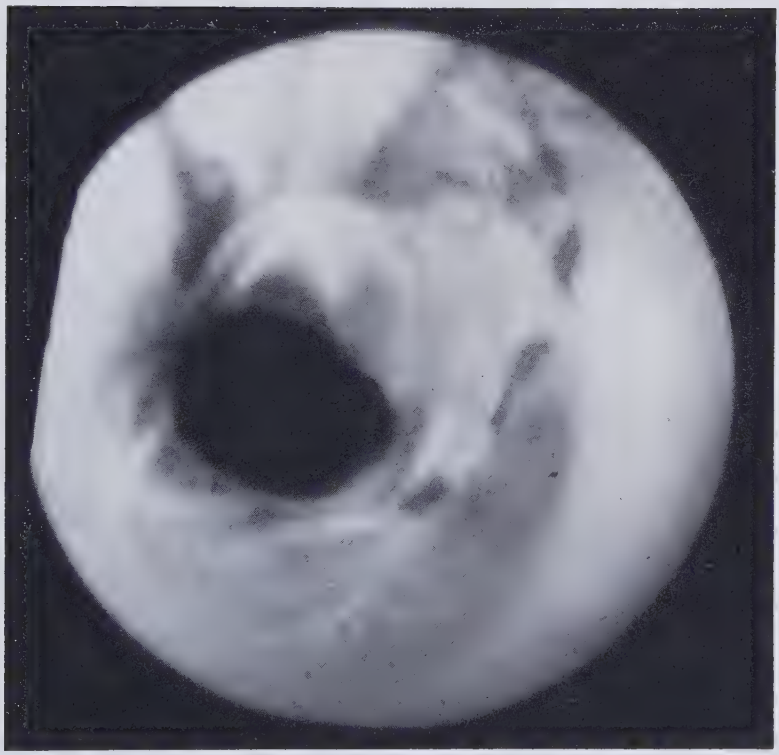

Figure 4. Venous valve in the greater saphenous vein. The endoscope is introduced in the direction of the bloodflow. An increasing central venous pressure provokes closing of the valve.

Figure 5. Remaining plaques and intima flaps in the external iliac artery after semi-closed thrombendarterectomy.

Table 2. *46 Vascular Endoscopies (on 45 Patients, between October 1, 1968 and November 30, 1972)**

ENDOSCOPIES OF THE ARTERIAL SYSTEM	PER CENT	ENDOSCOPIES OF THE VENOUS SYSTEM	PER CENT
Aortoiliac segment	24 Per cent	Iliac segment	6.5 Per cent
Femoropopliteal segment	63 Per cent	Femoral segment	6.5 Per cent

*Technical failure among all cases: 15.3 per cent.

CLINICAL EXPERIENCES AND DISCUSSION

From 1968 to 1972 we have done 46 intraoperative vascular endoscopies on 45 patients with these clinical indications: checking for a patent lumen during arterial reconstruction (86 per cent, see Table 2). The semiclosed thromboendarterectomy in the femoropopliteal area represents, among all arterial procedures, those which most frequently needed an endoscopic examination of the operative result.

The technical failure rate in this study was 15 per cent. Technical failure means either no possibility of introduction of the endoscope or insufficient intravascular view. In three patients using the rigid instrument the popliteal artery was too small (less than 6 mm. in diameter) for the introduction of the instrument. Since 1971 we have a fiberoptic endoscope with a smaller diameter available but we have not been confronted with this problem again. There arose no difficulties in placing the endoscope in the right position for the aortoiliac segment and the venous system. Kinking of the iliac arteries never caused a technical problem since we were using a flexible endoscope. Insufficient intraluminal vision was nearly always caused by incomplete bloodstream interruption or by insufficient perfusion with inadequate rejection of collateral inflow via nonclamped branches. Both technical failures can be eliminated by correct clamping, balloon vessel blockage or by an increase of the perfusion pressure. In some cases of venoscopy it was almost impossible to replace all the blood by saline solution. We see the reason for this phenomenon in a high volume capacity of the internal iliac vein system. In such situations endoscopy must be given up to avoid a cardiac overloading syndrome.

In 90 per cent of the reported cases it was possible to achieve an optimal visual verification of a patent lumen. For documentation the Hopkins Optical system of the rigid endoscope offers better optical conditions than the Fiberoptic system of the flexible instrument. On the other hand, for routine use the flexible endoscope always guaranteed a sufficient view. The advantages of the flexible endoscope are mainly technical ones (small diameter, better introduction into small vessels, especially in the femoropopliteal segment). In cases in which a high perfusion rate is necessary (iliac vein endoscopy), the rigid instrument has clear advantages due to the greater diameter of the perfusion channel.

Complications of vascular endoscopy using the technique presented

Table 3. *Complications of Vascular Endoscopy*

Mechanical lesions to the vascular wall
Perforation or dissection
Distention damage by uncontrolled pressure perfusion
Lesion to venous valves
Infection
Local bacterial contamination
Septicemia
"Overloading syndrome" by high infusion rate

above are remarkably few (Table 3). The most important complication was infection, either local or general. Initially we used a semiclosed perfusion system in which an unsterile pressure system was connected with a sterile perfusion solution. This was the reason for bacterial contamination of the saline solution in four cases, three of them followed by septicemia. This complication was completely eliminated by the use of a closed perfusion system similar to the Fenwall pressure infusion apparatus. Since changing to this system (1970) we have not seen further infections after vascular endoscopy.

Mechanical lesions of the vascular wall (such as perforation, dissection, or distention damage by uncontrolled pressure irrigation) can easily be avoided by gentle application of the endoscope.

In our patients there was no case of perforation. In the first year we registered four cases with mechanical distention damage to the disobliterated femoral artery due to uncontrolled pressure perfusion. In these

Figure 6. Endoscopic control after disobliteration of the iliac vein. Interruption of blood flow by clamping of the deep and superficial femoral veins and balloon blockage of the inferior vena cava. Continuous perfusion under controlled hypertension (50 mm. Hg) to prevent collateral inflow of blood.

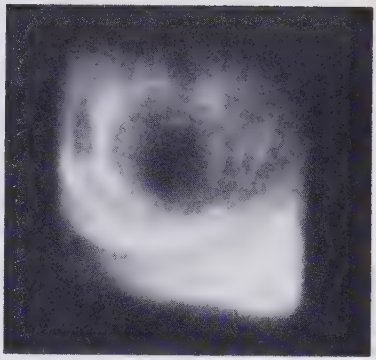

Figure 7. Complete disobliteration of the superficial femoral artery with the ringstripper. Inspection has been done with the fiberoptic instrument.

cases there was a rupture of the suture lines some days after the operation. Since that time we have used a pressure recording instrument with optimal results.

The danger of the circulation overloading syndrome caused by a high perfusion rate must be taken into consideration, especially when iliac vein endoscopy requires a high perfusion volume to clean up the vessel segment. The perfusion rate should never exceed 250 ml. per min. If under these conditions no clear view can be achieved the endoscopy should be given up.

In venoscopies damage to the venous valves can be avoided by cautious introduction and handling of the endoscope and by its introduction in the direction of blood flow.

In comparison to the other conventional intraoperative control methods (see Table 1), vascular endoscopy possesses some remarkable advantages with respect to diagnostic value:

The three conventional intraoperative control methods (flow control, palpatory lumen exploration by ring strippers, examination of the removed occluding cylinder) allow only rough information about vessel patency. Small remaining intima flaps or clots cannot be discovered by these methods.

Intraoperative angiography offers only a one-dimensional picture of the disobliterated vessel whereas endoscopy is able to give a complete three-dimensional view. Also, small intimal flaps can be seen regularly by endoscopy but only exceptionally by angiography.

Vascular endoscopy is done on open vessels. In contrast to intraoperative angiography there is no waiting time; a secondary correction of the lumen can be done immediately. This is not the case in intraoperative angiography. On the other hand, angiography is the diagnostic method of choice in all grafting procedures and in those at the level of small vessels (diameter below 6 mm). In these situations vascular endoscopy usually offers no diagnostic alternative except in the retrograde control of high level transplantation anastomosis if a more distal vascular segment is opened simultaneously.

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Innovative Applications of Extra-Anatomic Reconstruction

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In recent years a variety of limb salvage operations has evolved which does not follow classical lines of arterial reconstruction.

Since the original contributions of Lewis,[18] McCaughan and Kahn,[25] Blaisdell and Hall,[3] Louw,[19] and Vetto,[35] it has been proved that arterial grafts are not required to pursue the "normal" anatomic arterial pathways, but may indeed be implanted in seemingly unorthodox routes and still be quite successful. Most of these grafts follow a largely subcutaneous course. The most popular graft of this type currently in use is the axillary-femoral bypass graft. This originates in the first or second portion of the axillary artery and ends in the common, superficial, or deep femoral artery, either unilaterally or bilaterally. If one iliofemoral system is patent, another popular method is the suprapubic, subcutaneous, crossover, femorofemoral bypass graft. A modification of the latter is one extending from a patent external iliac artery and crossing over to a contralateral femoral artery, utilizing a combined retroperitoneal and subcutaneous route. A fourth type of extra-anatomic approach is one which utilizes the obturator foramen as a pathway to bypass the groin area when it is not available, usually as a result of infection.

Further variations include the less commonly employed axillary-popliteal bypass, the crossover femoropopliteal bypass,[13] the splenofemoral bypass,[20, 37] and the extraperitoneal thoracic-aorta to femoral artery bypass.[2] The latter two of these have been virtually abandoned with the advent of the "subcutaneous" grafts and are mentioned out of historic interest only. The discussion which follows will be centered on the three major groups of bypasses: the axillofemoral, the femorofemoral, and the obturator foramen approach.

Indications for the Extra-Anatomic Approach

Leg-threatening iliac and aortoiliac arterial occlusive lesions have classically been treated by endarterectomy or aortoiliac and/or aortofe-

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Table 1. *Indications for Use of Extra-Anatomic Arterial Reconstruction Procedures*

A. Conditions contraindicating major surgery
Advanced age and debilitation
Significant cardiopulmonary disease
Limited life expectancy
B. Need for a clean field
Infection of an aortic suture line
Infection of a groin anastomosis
Colostomy
Intra-abdominal sepsis
Frozen pelvis secondary to tumor or radiation
C. Technical problems
Multiple previous intra-abdominal procedures
Saddle or iliac embolus or thrombosis not correctable via a groin incision
Occlusion of one limb of a bifurcated graft

moral bypass procedures. The results of these operations have been uniformly good. Many patients, however, are unable to tolerate operations of such magnitude, though the advantages of restoring full aortic pressure to a distal patent circulation are obvious. These patients need not, however, be resigned to a major amputation which, in itself, carries a formidable risk.

An outline of the more common situations precluding major abdominal surgery is presented in Table 1. In such situations, an approach other than the classical ones should be sought to revascularize ischemic lower extremities. It is apparent that the "indications" for the extra-anatomic approach are really just contraindications to the anatomic method. The patient who is included in group A is unlikely to survive an operation of the magnitude of aortoiliofemoral reconstruction, or may be suffering from end stage malignancy and all that is required is a temporary procedure. If the axillofemoral or femorofemoral graft were to fail after a period of time, it is conceivable that the medical contraindications to major surgery may no longer be as severe, and at that time the classical reconstruction could be performed.

Group B represents the need for a "clean field." Infection, in particular, requires removal of pre-existing foreign body and scrupulous avoidance of the contaminated area with a new prosthesis. These previously unsalvagable patients are now frequently benefited by the new techniques available.

The technical problems of Group C may occur alone or in combination with the other two groups. An example would be the patient who has sudden occlusion of the aorta or iliacs following an acute myocardial infarction and cannot be revascularized by retrograde Fogarty thromboembolectomy because of severe pre-existing atherosclerotic changes in the iliac vessels. Similarly, a previously functioning bifurcation graft may have one or both limbs thrombose under like circumstances with resulting severe ischemia.

In all of the above situations, the availability of a secondary, less major, alternate pathway for blood flow is invaluable.

AXILLOFEMORAL BYPASS

Lewis[18] deserves credit for first utilizing the vasculature of the upper extremities to supply blood to the lower half of the body. Faced with a ruptured abdominal aortic aneurysm plus dissection of the thoracoabdominal aorta precluding adequate inflow, he utilized a nylon graft anastomosed end-to-end to the left subclavian artery to restore flow to the proximal portion of an aortic homograft. Within a few years, reports by Louw,[19, 20] and Blaisdell and Hall[3] appeared and the concept of the axillofemoral bypass was born. Numerous series and individual case reports[4, 14, 24, 27, 29] have since appeared to supplement the early observations and establish this somewhat unusual approach within the armamentarium of the vascular surgeon.

Technique

In even the most advanced cases of ischemia, if the limb is still viable, the deep femoral system is almost invariably patent. This simple fact is responsible for the necessity of considering revascularization before embarking on amputation. When an axillofemoral bypass is to be performed, the patient is placed supine on the operating table with the "donor" arm extended on an arm board. Needless to say, intravenous pathways and blood pressure manometers should be placed on the contralateral arm. The arm, axilla, chest, abdomen, groin and thigh are then prepared and draped. Since the operation is all subcutaneous except for the proximal subpectoral portion, light general (possibly supplemented by local) anesthesia can be used.

The axillary artery is approached through a subclavicular incision with splitting of the fibers of the pectoralis major muscle. It is our custom to cut all of the medial portion of the pectoralis minor muscle insertion on the coracoid process in order to afford wider exposure. Either the first or second portion of the axillary artery is then cleared and small branches are divided. It is important to attach the graft to the first or second portion of the axillary artery rather than the third, for two major reasons. Motion of the arm will cause tension and perhaps kinking or bending of the graft if the third portion is used. Since the axillary artery is well fixed as it emerges from under the clavicle, this is not a problem if the proximal two thirds are used. Also, in order to make the graft anastomosis at a sufficiently acute angle and still prevent angulation, it should follow a gentle curve toward the midaxillary line. It is also important to avoid excessive tension on the graft, which can cause kinking and turbulence in the axillary artery, leading in turn to thrombus formation near the anastomosis site.[24]

The groin vessels are then approached through a standard vertical incision if infection does not supervene. The two wounds are then connected in the following manner: proximally, a tunnel is made with a blunt instrument or finger from the subclavicular wound under the pectoralis major muscle to emerge subcutaneously in the anterior or midaxillary line. The tunnel is then continued subcutaneously in this line until it reaches the level of the iliac crest where it then gently curves anteriorly

to reach the groin incision. In order to maintain perfect alignment, prevent constriction, and avoid twisting the graft when it is brought through the tunnel, it is desirable to make one to three counter ("leapfrog") incisions along the way. Avoidance of the flare of the costal margin, which can cause kinking when the patient sits or bends,[24] and also too posterior a position, so that recumbency produces a pressure on the graft, requires this strict lateral placement.

Finally, the anastomosis in the groin is done in the usual end-to-side fashion, either to the common and superficial femoral arteries or, if the superficial femoral is occluded, as an onlay patch over the deep femoral artery. Endarterectomy of the deep femoral orifice may be added to this latter method if necessary. The necessity for providing adequate outflow (which is often solely via the deep femoral system) cannot be overemphasized. A drain is sometimes placed in the subclavicular wound where a significant potential space exists, but the groin wound should be carefully and firmly closed in layers without draining. This procedure is diagrammatically depicted in Figure 1*A*, and a postoperative arteriogram is shown in Figure 1*B*.

A somewhat different approach may be necessary if infection is the indication for axillofemoral bypass grafting. It is not uncommon for both the superficial and deep femoral artery origins to be jeopardized when the limb of an aortofemoral graft becomes infected at the groin level, for the distal anastomosis is usually performed at or near the common femoral artery bifurcation. Such a case is illustrated in Figure 2*A*. This 60 year old white male had undergone aortofemoral bypass grafting 2½ years previously for intermittent claudication. Following discharge from the hospital, he did well until an episode of diverticulitis of the sigmoid colon ended in perforation and abscess formation along the left limb of the graft. Right transverse colostomy controlled the inflammatory process in the bowel, but the infection quickly spread downward to the groin where anastomosis disruption and bleeding ensued.

In order to avoid the contaminated area and yet preserve the patency of both the superficial and deep femoral arteries, the following was done (Fig. 2*B*). The infected left groin area was draped out of the operative field. Next, the axillary-deep femoral bypass graft was inserted via carefully prepped incisions with the graft tunnelled subcutaneously and *lateral* to the anterior superior spine of the iliac crest, brought medially under the sartorious muscle, and anastomosed in a clean field to the deep femoral artery. The bifurcation of the common femoral artery was lowered by dividing the deep femoral artery distal to the "old" graft anastomosis and anastomosing the transected distal stump of the deep femoral artery to the side of the superficial femoral artery. Ligation of the proximal stump of deep femoral artery, as well as of the superficial femoral artery proximal to the new anastomosis (all in a clean field), prevented backbleeding into the contaminated groin area above.

Next, the abdomen was entered via a clean field, and the origin of the infected left limb of Dacron bifurcated graft was transected and oversewn proximally and ligated distally, after noting that the proximal portion of the limb did not appear to be infected. The abdominal and ax-

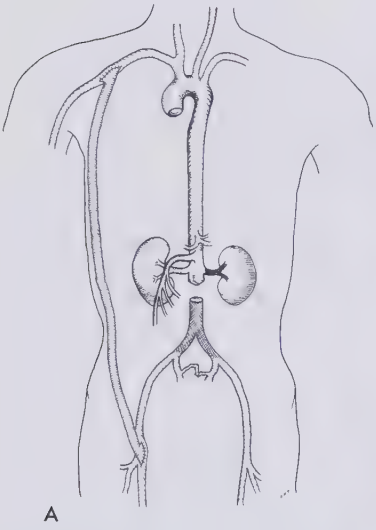


Figure 1. *A*, Diagram of an axillo-femoral bypass graft in a debilitated patient with much scarring in the terminal aorta and iliac arteries. *B*, Arteriogram of such a patient, demonstrating axillo-femoral bypass.

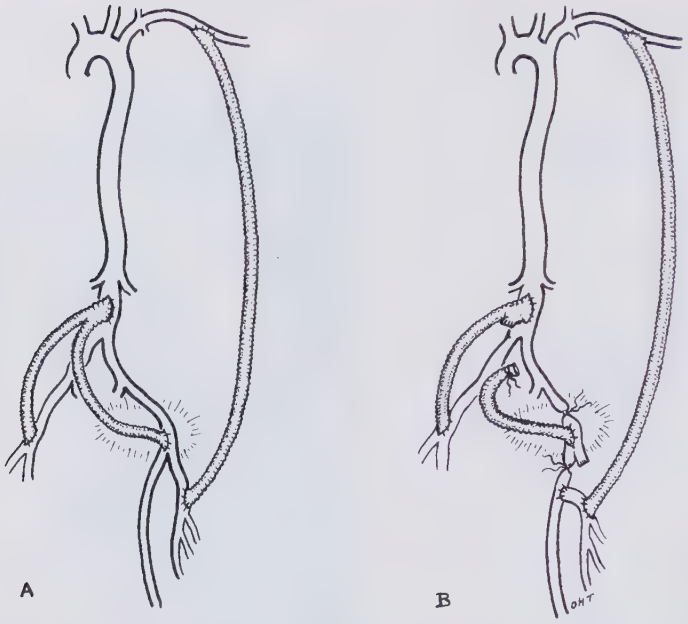


Figure 2. *A*, Diagram of patient with infected distal portion of left limb of aortofemoral Dacron graft showing left axillary to *deep* femoral bypass graft via a clean field. *B*, Diagram of same patient showing lowering of the common femoral bifurcation (via a clean field) and division prior to removal of the origin of the left limb of the distally infected graft.

illofemoral bypass incisions were then closed under sterile conditions. Return to the contaminated groin area now allowed removal of the infected limb of graft from below, thus minimizing the likelihood of intraabdominal contamination. After removal of the foreign body, slow but complete secondary healing of the groin area ensued. Meanwhile ankle pulses were maintained by way of the rather circuitous new route and no subsequent infection occurred.

Both of the previous descriptions have centered on the patient with severe ischemia of only one limb. In the event that both lower limbs are threatened, an axillary bilateral femoral graft can be performed.[5, 24, 28] It has been suggested that this provides greater outflow for the main body of the graft, thus ensuring longer patency. This supposition, however, is without significant evidence, but the procedure of attaching a side arm graft at or near the common femoral anastomosis as a bypass (again subcutaneously) to the opposite groin is probably simpler and less time-consuming than the alternative of creating a separate and distinct axillofemoral graft on the contralateral side.

An unusual case has been described by Jackson and Ward[16] where severe ischemia of the right leg was present in a patient with significant but asymptomatic stenosis of the contralateral iliac artery and ipsilateral subclavian-axillary arterial system. This was treated by axillofemoral bypass from the *left* axillary to the *right* femoral artery with good results.

Results

The usual reason for choosing this extra-anatomic approach for revascularization over the standard direct methods is to avoid the mortality and morbidity attending the latter operations. The results reported in the literature as well as in our own series show achievement of this goal. Mortality rates of under 10 per cent are found, and this is certainly acceptable considering the poor general preoperative condition of the patients. In fact, this is significantly less than the mortality rate of major amputation for gangrene in patients of this age group. When death does occur in the postoperative period, it is usually due to associated cardiopulmonary disease and/or accompanying sepsis and hemorrhage.

Morbidity is minimal since neither thorax nor abdomen is entered and postoperative pain is slight. The patient is allowed to eat on the day of surgery and may be ambulated the next day. Infection in these grafts is only infrequently seen.[25] Ischemia of the "donor" upper extremity is similarly rare. Hand or arm pain on exercise (arm claudication) has been reported by Sauvage and Wood[28] but was far from disabling. We have had two cases of late occlusion (18 months and 2 years after bypass) of the axillary artery, one of which required thrombectomy, and both were associated with occlusion of the grafts.

Because of the relative newness of this procedure and the lack of sufficiently large series, long term patency rates are difficult to determine. The two largest series are those of Blaisdell et al.[4] with 41 grafts and Mannick et al.[24] with 46 grafts. In the former, the 2 year patency rate was 80 per cent if the distal circulation was normal and only 50 per cent if an outflow vessel was occluded. With follow-up of up to 5 years, Mannick reported a 72 per cent overall patency rate, and in fact most of the failures occurred early since of 27 patients studied for 1 to 5 years, only 3 suffered late occlusion of the graft. In our personal cases 2 year patency has approached 75 per cent with most occlusions occurring later than 6 months postoperatively.

When occlusion does occur, it is frequently seen when the patient awakens in the morning. Since the graft is subcutaneous, compression by the patient sleeping upon it is thought to play a significant role. Wearing constricting clothing (e.g., belts or girdles) and sleeping on the involved side should be avoided. It should be emphasized that if occlusion does occur, an attempt at thrombectomy of the graft should be done since successful long term patency can often be restored (Mannick[24]—7 of 10 thrombectomies successful). Intraoperative arteriography at the time of thrombectomy will often yield the cause for occlusion (i.e., twisting or kinking or constriction of the graft if early occlusion, stenosis of the distal anastomosis if late), and appropriate steps may be taken to prevent reocclusion.

Louw[20] and Stipa[31] among others, have suggested that the saphenous vein (or cephalic or basilic veins) be used for this bypass instead of Dacron, the rationale being that it is a long graft comparable to femoropopliteal bypasses and, therefore, greater long term patency can be expected if autogenous material is used. We feel that there are several reasons for *not* using the saphenous vein. First, the vein will at best be

considerably smaller than an 8 or 10 mm. Dacron graft and thus be incapable of delivering as high a flow rate. Second, since these patients are poor operative risks, operating time should not be extended as it must be to harvest the vein. Third, the vein may well be required for the treatment of distal disease. Finally, Mannick[23] has documented flow rates in a Dacron axillofemoral graft of 550 ml. per min. if both the superficial and deep femoral arteries are patent and 120 ml. per min. if only the deep femoral is patent. This latter figure represents near the *maximum* flow that can be achieved with a femoropopliteal bypass and thus the situations are not really comparable. Similarly, Blaisdell[4] has shown that "free flow" through the graft after axillary anastomosis approaches the entire cardiac output. Documentation of greater long term patency with saphenous vein axillofemoral grafts is necessary if the above mentioned factors are to be obviated.

FEMOROFEMORAL GRAFTS

Even before the advent of axillofemoral grafting, it was recognized that blood could be "borrowed" from one nonischemic leg to supply the opposite symptomatic extremity. McGaughan and Kahn in 1960[25] described a graft from the left external iliac artery to the contralateral popliteal artery and Vetto[32] in 1962 reported 10 cases where femorofemoral crossover-grafting was used. Several relatively large series with surprisingly good results have followed, despite the fact that aortoiliac occlusive disease is usually bilateral rather than unilateral in distribution.

Technique

Ease of exposure, and proximity of the two incisions make femorofemoral bypass even simpler to perform than axillofemoral bypass. Thus, local anesthesia is often all that is required, a decided benefit in the seriously ill individual. The abdomen, both groins, and both upper thighs are prepared and draped. Aortography is useful but not essential in determining the adequacy of the "donor" iliofemoral system preoperatively, as an adequate femoral pulse on the nonsymptomatic side may be taken as evidence of sufficient inflow. A vertical incision is then made in the groin over the common, superficial, and deep femoral arteries of the affected limb. This should be done prior to exposing the inflow side in order to assess the availability of an outflow tract. As with the axillofemoral method, endarterectomy of the deep femoral orifice or onlay of the graft to widen the deep femoral may be necessary to achieve such outflow. The other groin is then explored and a subcutaneous tunnel is created by blunt dissection between the two wounds just cephalad to the pubis.

The donor common femoral artery is then opened on its medial aspect. If necessary, endarterectomy or other reconstructive procedures can be performed in this area.[1] An end-to-end side anastomosis is then performed using an 8 or 10 mm. knitted Dacron graft. Although an attempt is generally made to provide an acute angle for the takeoff of the graft and thus diminish turbulence which can lead to thrombosis,[33] this

has not proved essential,[36] and continued long term patency is achieved despite a "right angle takeoff."

The graft is then brought through the tunnel and anastomosed to a suitable arteriotomy in either the common or deep femoral artery of the ischemic limb, as previously described (Fig. 3). The wounds are then closed carefully in layers without draining. Since this is a short graft and for the reasons described above under axillofemoral grafting, there is usually no need to use the saphenous vein for this bypass, although this has been described.[10, 11, 17, 38] One possible indication may be when chances of infection are great.[26]

If iliofemoral crossover bypass is to be done (especially in the case where significant disease exists in the donor external iliac artery), then the above procedure is modified by exposing the contralateral iliac artery via a retroperitoneal approach. The graft will then pass retroperitoneally, suprapubically, and deep to the inguinal ligament (Fig. 4). Occasionally, in order to improve the outflow, it is necessary to continue the graft down to the popliteal artery, as has been described by Ayvazian et al.,[1] Goetz and Lord,[13] and Foley et al.[11]

Results

Although great concern has been expressed that one iliofemoral system would be unable to supply blood for both legs and that a "steal" from the donor limb might develop,[11, 38] only sporadic reports of such occurrences have appeared.[11, 34] Parsonnet et al.[27] have rightfully pointed out that during cardiopulmonary bypass, the entire cardiac output may be supplied by one common femoral artery. Ehrenfeld and his colleagues, in an experimental study, showed that the effect of the bypass was to double the flow in the proximal donor artery rather than steal from the donor extremity. Sumner and Strandness[32] have reported that a steal may be detected by measuring ankle pressures and blood flow, particularly on exercise and when the donor iliac is stenotic. However, if no significant donor obstruction exists, no steal will develop, and at rest there is no apparent steal even if the donor iliac is stenotic.

Trimble et al.[34] did encounter ischemic symptoms in the donor limb in two of their patients. Using an experimental model they concluded that this problem will develop only if the peripheral resistance (i.e., distal occlusive disease) is greater in the donor limb than in the recipient limb.

As expected, the mortality and morbidity associated with this procedure are as minimal as the required operative dissection. What were not initially anticipated were the excellent results so far observed. A survey of the reported series[4, 6, 11, 17, 24, 27, 32, 34, 36] reveals that of 212 grafts placed, there was an initial patency rate of over 91 per cent. Even more surprising is the remarkably low attrition rate with longer follow-up (in many cases approaching 5 years), since success continues in 88 per cent.

It was thought that progression of disease in the donor iliac artery would lead to many late failures but this has not been the case. In fact, the results suggest that some protective influence may be exerted on the donor iliac artery by the presence of a crossover graft. Vetto[36] has theorized that increased linear blood flow causes a decrease in lateral ves-

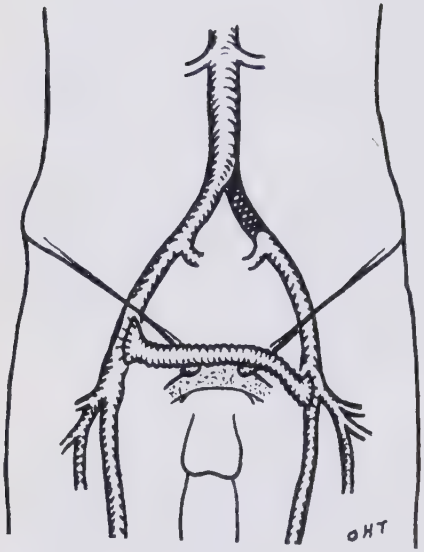
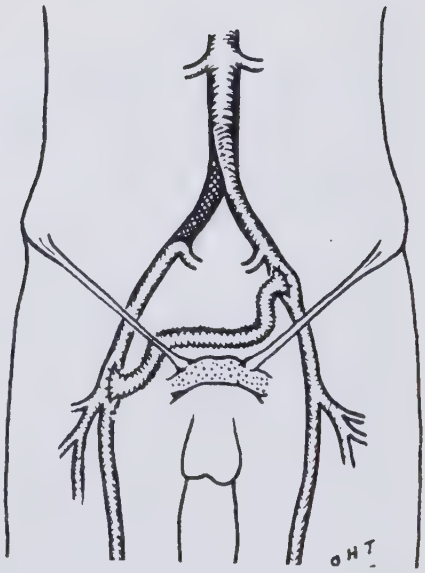


Figure 3. Diagram of the femorofemoral, subcutaneous, crossover, bypass graft.

Figure 4. Diagram of the iliofemoral, retroperitoneal, crossover bypass graft.



sel wall tension and thus less trauma and subsequent atherosclerosis. Mannick[23] suggests that increased flow in the donor iliac artery prevents the deposition of fibrin and platelets, with resulting prolongation of patency.

Because of these excellent long term results, which compare very favorably with aortofemoral bypass, the indications for femorofemoral grafts have been expanded by several authors[4, 6, 23, 36] to include patients with claudication only. Indeed, Brief and his associates[6] now believe that this is the operation of choice for unilateral aortoiliac occlusive disease. Until more complete 5 and 10 year follow-up data accumulate, we prefer to withhold final judgment on this point.

BYPASS VIA THE OBTURATOR FORAMEN

Sepsis is the nightmare of the surgeon who undertakes reconstructive surgery of major arteries. In the past this complication has almost inevitably led to major arterial hemorrhage and hence to death of either the patient or a considerable portion of him.[29]

The above eloquent quotation is only too painfully true and underscores the necessity for alternate pathways in providing arterial reconstruction in the face of infection, particularly in the groin area. Since Shaw and Baue's original contribution,[29] bypass via the obturator foramen has provided just such an avenue of escape from the horrors of an infected prosthesis.[8-10, 12, 15, 21, 30] The time-honored surgical principle of dealing with infection complicated by a foreign body, i.e., removal of the foreign body and closure by secondary intention, can thus be followed with preservation of the vasculature. The obturator foramen bypass is a considerably more formidable undertaking than axillofemoral or femorofemoral bypass, but its indications are specific and limited to an infected or unapproachable groin (e.g., tumor, radiation necrosis). Because of this, its use has been considerably less frequent and the morbidity and mortality associated with it are significantly greater.

The technical aspects of this procedure are well described by Shaw and Baue[29] and beautifully illustrated by Guida and Moore.[15] The proximal portion of the operation may be done either transperitoneally or retroperitoneally. The common, external and internal iliac vessels are mobilized as well as the affected limb of the aortofemoral graft, if one is present. Care must be taken to avoid the ureter in this area. If possible, the obturator artery is identified as it originates from the internal iliac but occasionally it will arise from the inferior epigastric artery. Next, an incision is made along the medial aspect of the upper thigh directly over the superficial femoral artery but a safe distance distal to the contaminated groin. The adductor longus muscle is retracted laterally to expose the adductor brevis with the obturator nerve lying upon it. Blunt dissection superiorly in this plane between the two muscles leads to the obturator foramen. A tough, membranous fascia overlies this foramen within the pelvis and it must be incised sharply from above in order to create a wide enough opening. The incision is made in the anteromedial aspect, care

being taken to avoid the neurovascular bundle which lies superiorly. A tunnel is then developed between the two clean wounds to accommodate the bypass graft.

A graft may then be connected proximally to the aorta or iliac artery, or a previously placed (but uncontaminated) graft may be brought through the tunnel to anastomose to the mid or distal superficial femoral artery or to the popliteal artery, depending on the outflow situation. After appropriate closure of these clean wounds, the infected groin can be approached, where ligation and debridement are performed above and below to effect hemostasis. The groin wound should then be left open to allow granulation and secondary closure.

Limb salvage as a result of this procedure can be expected if the infection can be controlled and if reinfection in the new graft is avoided. Meticulous attention to detail is necessary in order to prevent cross contamination. No large series of long term results exists, as this bypass is infrequently performed. However, numerous case reports indicate that even difficult complications can often be overcome using the obturator foramen bypass route.

SUMMARY

Over a decade of experience has proven the value of certain unconventional, extra-anatomic arterial reconstructive procedures in rescuing the lower extremity from gangrene. Numerous investigators have demonstrated that advanced iliac artery occlusive disease does not necessarily require large magnitude, direct aortoiliofemoral reconstruction in order to salvage the ischemic leg.

As alternative operations, the axillofemoral bypass, the femorofemoral bypass, and their modifications are valuable in patients who are debilitated, who have intraabdominal sepsis or scarring, or who for any reason have unapproachable aortoiliac vessels. The long term patency rate of these procedures, especially the femorofemoral crossover bypass, is highly acceptable.

The transobturator foramen bypass has a special usage in revascularizing the ischemic lower limbs of patients who have infection, tumor, or other disease in the groin area.

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Amputation and Rehabilitation for Severe Foot Ischemia

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The patient with peripheral vascular disease who is facing irretrievable loss of a portion of his lower extremity has a unique problem. The surgeon will not encounter the same relatively easy problems that exist in other disease states requiring amputations. The patient with peripheral vascular disease is generally in the geriatric age group, and in addition to an ischemic foot, may have lessened muscular strength, lowered cardiorespiratory reserve, generally less agility and balance, probably poor vision, perhaps impaired hearing, and usually lessened motivation for rehabilitation. In addition, and most important, the surgeon will most likely perform the amputation at a level that may have marginal blood flow.

The modern attitude toward amputation surgery in the patient with peripheral vascular disease spurns the "safe" approach, that is to say the above-knee level of amputation, in favor of the most distal amputation level (preferably below the knee) consistent with wound healing. The literature of the last 15 years reveals a very definite change in attitude toward amputation in the patient with the ischemic foot. Ottman and Stahlgren,[10] surveying the experience at Philadelphia General Hospital from 1955 to 1960, found that in 323 geriatric patients with amputations performed because of avascular lower extremities, above-knee amputations were performed in 307 of the patients and below-knee amputations in only 10. In the years between 1962 and 1967, Lim et al.[8] found that of 95 amputations performed because of vascular disease, 55 were below the knee, whereas 40 were above the knee. In this study, only 17 per cent of the below-knee procedures had to be revised to above-knee amputations.

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As one progresses to the more contemporary literature, one finds, for example, Sarmiento noting that since 1954 the percentage of below-knee amputations has risen from a low of 30.7 per cent per year to 83 per cent per year.[7] The percentage of overall rehabilitation successes rose from 24 per cent to 61 per cent.

Most general surgeons and many vascular surgeons feel relatively comfortable performing amputation surgery. However, to this group, an amputation is generally an admission of a surgical defeat. The surgeon has not been able, sometimes by the most sophisticated surgical procedures, to provide sufficient blood flow to the extremity to prevent its distal death. The patient has a painful, necrotic, sometimes odorous extremity, that is incapable of comfortably bearing weight or providing ambulation. The patient is, in short, crippled.

The surgeon, whether he be a general or an orthopedic surgeon, who approaches this patient with the expectation of providing optimal patient rehabilitation, may well regard the amputation procedure as a surgical triumph when it is the *first step in rehabilitation* leading to renewed comfortable ambulation. Independent ambulation in the older amputee, secondary to peripheral vascular disease (which is usually a generalized process), is admittedly difficult. It requires performance of an amputation producing as long a healed stump as is possible, consistent with the level of the disease process; adequate preprosthetic treatment; the application of a proper prosthesis; and an adequate postprosthetic rehabilitation program. Ideally, this whole program should be under the general direction of the amputating surgeon. However, since medical practice today is generally far from ideal, compromises usually have to be made. It is the opinion of the authors and others[1, 2, 6, 7, 11] that the area in need of the greatest improvement is the initial step, namely the performance of an amputation which yields a healed stump (usually with preservation of the knee joint) that is capable of comfortable and functional mating with a prosthesis.

There are few locations in this country today that do not have access to an amputee clinic or a rehabilitation center, so that the rehabilitation phase may be delegated to these facilities. However, the surgeon should be aware that referral to such clinics should be made early—in most patients, as soon as the sutures are removed. If the patient arrives at the amputee clinic or rehabilitation center before general debilitation has occurred, before joint contractures have become fixed, and before stump edema progresses to soft tissue fibrosis (Fig. 1), rehabilitation moves along at a rate that is more likely to maintain patient motivation, interest, and subsequent successful ambulation.

AMPUTATION LEVELS

In patients with disease states other than peripheral vascular, the surgeon may have a choice of several amputation levels. Gangrene of a toe or several toes may indicate to the surgeon simple amputation of the involved toes. However, healing may be delayed or produce a locally in-

Figure 1. Stump edema due to poor postamputation bandaging.

fected unhealed area, which subsequently requires higher amputation. It is the opinion of the authors that it is better to resect at the transmetatarsal level for this limited involvement, providing the patient meets the requirements promulgated by Pedersen and Day.[11] These authors indicate that in the patient with dry gangrene, involving one or more toes, with no infection present in the foot, the foot itself warm to the touch, and without "rest pain," a transmetatarsal amputation will usually heal. A crushed foot in a young adult may provide sufficient tissue to produce a well-healed Syme's amputation stump, but a Syme's amputation will not heal in many patients with peripheral vascular disease. This amputation demands good to excellent circulation in the heel flap. Some surgeons have reported success with a two-stage Syme's amputation for foot ischemia secondary to peripheral vascular disease, but these amputations have not generally been successful in most surgeons' experience.[9, 13] If the gangrenous process involves more of the forefoot, is infected, and "rest pain" is present, the surgeon is well advised to go to a definitive below-knee level (Fig. 2).

TRANSMETATARSAL AMPUTATIONS

If the cited requirements are present, this level of amputation will usually succeed in two of every three patients.[12] This amputation level does require careful attention to detail, and most important, gentle handling of tissue to prevent further local tissue necrosis. The incisions are planned so that the dorsal incision will be at the level of bone section and will remain relatively undissected from the deeper structures (Fig. 3).

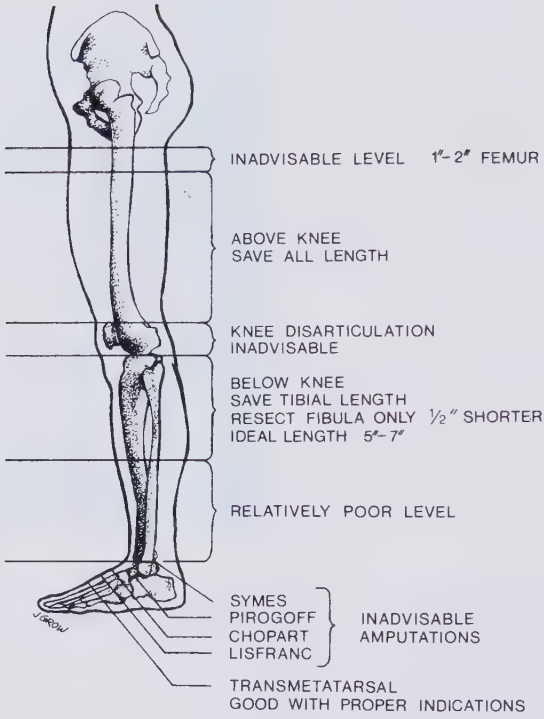

Figure 2. Amputation levels of opportunity in peripheral vascular disease.

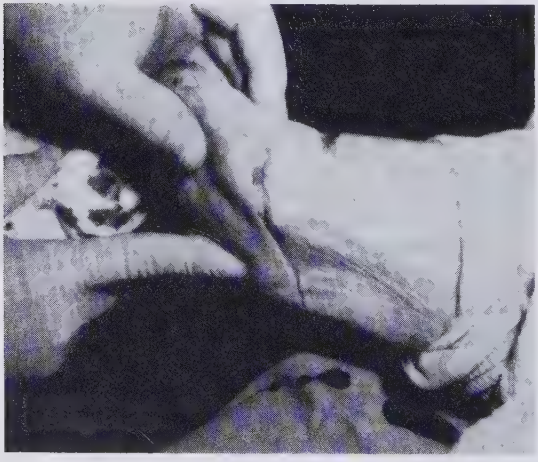

Figure 3. Incision for transmetatarsal amputation.

The toe extensor tendons are cut flush, or may be gently pulled down and sectioned high, so that avascular tendon tissue is not present in the area of incision. The metatarsal bones are then sectioned as atraumatically as possible, which usually requires a power saw or Gigli saw, and the bone edges are then smoothed.

The plantar flap is made long enough to turn up to meet the dorsal skin flap, and the flexor tendons are sectioned at the level of bone section or slightly proximal. All bleeding points are carefully ligated. The digital nerves are sought for and gently stretched distally, and sectioned as proximally as possible. The skin is closed as atraumatically as possible. If desired, a well-padded immediate plaster rigid dressing can be applied to minimize or prevent wound edema. When such a dressing is applied, it is important that the foot be maintained at a right angle to the leg. If wound edge necrosis appears. it is not necessary to consider revision immediately, since, in most instances, healing will occur if the surgeon is sufficiently patient. Fortunately, this amputation level requires very little in the way of prosthetic replacement, usually only a firm leather shoe with a steel shank reinforcement, and a toe filler.

PARTIAL FOOT AMPUTATIONS

It has been common knowledge that in most instances the partial foot amputations, including Chopart's, Lisfranc's, and Syme's, are usually not good amputation levels when the diagnosis is peripheral vascular disease. When "rest pain" is present in the foot, and the gangrenous area is infected, the survival, with healing, of an amputation distal to the below-knee level is questionable. Even if a patient were fortunate enough to heal with a Lisfranc or Chopart level amputation, he will usually have difficulty ambulating comfortably with the presently supplied type of prosthesis. From the practical standpoint, either a transmetatarsal amputation or a standard (5 to 7 inches) below-knee amputation is indicated in peripheral vascular disease. Even if the gangrenous process has approached to the midcalf area (Fig. 4*A*), a short below-knee stump can be salvaged in many instances, and will usually not require revision to an above-knee level (Fig. 4*B*).

THE BELOW-KNEE AMPUTATION

The knee joint is the most important structure during ambulation, and should be preserved in all possible instances.[11] In the upper extremity, the loss of the hand is the major loss to the patient; however, in the lower extremity, the major loss is not that of a foot, but rather that of the knee joint. Retention of the knee joint is of great value to the patient, even when he is not using his prosthesis. The functioning knee joint enables him to more easily turn over in bed, and makes transfer from bed to wheelchair easier. In ambulating with a below-knee prosthesis, the patient has better and more secure prosthetic control, with considerably

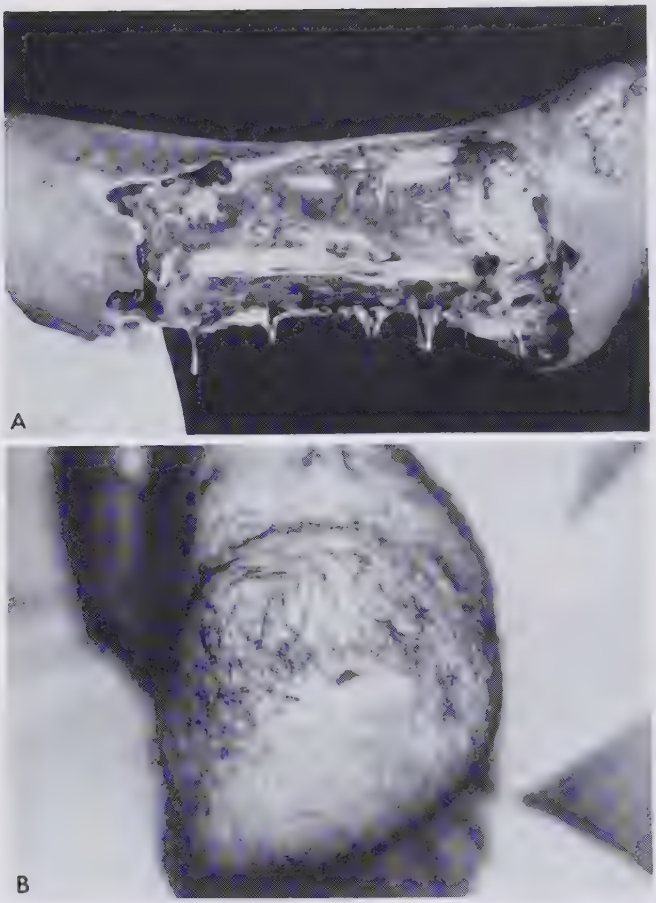

Figure 4. *A*, Diabetic gangrene of leg. *B*, Healed below-knee amputation stump.

less expenditure of body energy. A unilateral below-knee geriatric amputee with a healed, nonpainful stump will usually ambulate with a prosthesis, using only a cane. Even a bilateral below-knee amputee will, in the majority of instances, be rehabilitated to ambulation using prostheses, requiring the use of one or two canes (Fig. 5). It is highly important, therefore, to reorient our thinking processes to include preservation of the knee joint.

Many surgeons have, over a period of years attempted to elucidate the factors that would enable the surgeon to accurately predict the proper level of amputation. In peripheral vascular disease, the above-knee level has had many advocates for many years. However, those who have advocated a more conservative approach to amputation have not been silenced, and some of these were promoters of the below-knee level even before the development of the modern immediate postsurgical fitting technique. In 1944, Silbert[11] was recommending below-knee levels of

Figure 5. Patient with bilateral below-knee amputations and prostheses.

amputation for peripheral vascular disease, although admittedly espousing the open circular amputation over the closed method. His only contraindication to the below-knee amputation was in the patient with a recent thrombosis of the femoral artery with abrupt pain, and with no previous history of claudication. He voiced the opinion that an absent popliteal pulse was not a criterion for an above-knee amputation.

Considerable time has been devoted to the study of the pattern of arterial involvement in the arteriosclerotic obliterans patient, both with and without diabetes. A number of authors have noted that diabetes increases the arteriosclerotic involvement of the distal arterial tree. Haimovici[5] noted that in pure arteriosclerotic obliterans disease, a single artery in the below-knee area was involved in 65 per cent of patients, whereas 69 per cent of patients with diabetes and arteriosclerotic obliterans show involvement of all three arteries *below* the knee.

Strandness et al.[15] also noted that in diabetes there was a higher incidence of arterial occlusion in all three major arteries below the knee, but in the nondiabetic patient with arteriosclerotic obliterans, there was definitely *more proximal aortofemoral involvement*. Other authors have also noted that the involvement of the aortoiliac segment of the arterial tree was twice as common among nondiabetic arteriosclerotic obliterans patients as among those with diabetes.[3] These studies tend to show that diabetes mellitus produces more frequent and greater involvement of the arteries in the below-knee area, and therefore produces a greater incidence of gangrene in the foot, than is the case in the arteriosclerotic obliterans patient without diabetes. However, those patients without

diabetes, who have arteriosclerotic involvement, have a greater incidence of proximal occlusion, which may be of a sudden nature, and therefore are less likely to develop collaterals in sufficient time to prevent more extensive gangrene of the lower extremity. These facts rationalize what common observation has noted, namely that patients with gangrene secondary to diabetes are more likely to heal with a below-knee amputation than those who have arteriosclerotic obliterans without diabetes.

It is the considered opinion of those who have contributed to amputation surgery techniques in the past several years that at present, there is no nondestructive method of determining arterial blood flow that will, with certainty, provide the surgeon with the knowledge of which amputation level will heal and which will not heal. Subsequently, it has been found useful when selecting an amputation level to note that the patient who demonstrates adequate skin blood flow (after skin incision) at the level of section, will usually have sufficient healing ability to warrant the completion of the below-knee level of amputation.[1] A below-knee level of amputation retaining 5 to 7 inches of length yields an excellent stump for prosthetic use. However, it is unnecessary to measure the stump length so exactly; as a rule of thumb, if the amputation level is in the proximal portion of the middle third, a quite satisfactory below-knee amputation will result. Of course, gangrene which has advanced to just below this level may provide less than an ideal below-knee length. However, if the surgeon can preserve as little as 2 inches of below-the-knee length, with the patellar tendon insertion intact, the resultant stump will be adequate to fully function as a below-knee amputation.

Some patients with an ischemic foot may also have infected, ulcerated lesions of the extremity, or perhaps an unhealed infected partial foot amputation. These infected areas should be sealed away from the area of surgery, in order to minimize or prevent contamination of the operative wound. If the area of infectious involvement is only the foot, the placing of a large, sterile rubber glove over the foot and above the ankle joint will provide an impermeable cover for the contaminated area and allow a sterile preparation of the area of incision. If the infected area involves the lower third or half of the leg, the lower portion of the leg may be wrapped with Steridrape, again sealing the infected area away from the area of incision. The surgeon may work as close as 1 to 2 inches to such an ulcerated, infected area, and still maintain relatively sterile skin conditions at the incision level.

Long Posterior Flap

It has been a common observation that the below-knee skin circulation in peripheral vascular disease is usually greater in the posterior calf area than in the anterior shin area. Thus, when skin flaps are fashioned of equal length, one may find that the anterior lateral portion of the flap becomes necrotic (Fig. 6). The long posterior skin flap was apparently initially described by Ghormley,[4] but has been recently revived and advocated by Burgess.[1]

The anterior skin incision is made transversely, and is *not* appreciably dissected upward from the area of incision, thus minimizing loss of

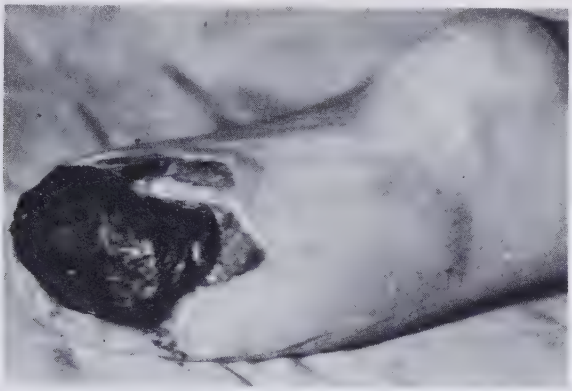

Figure 6. Gangrene of anterior skin flap.

skin blood flow. At the lateral and medial midpoint of the leg, the incision is carried distally to provide a long posterior skin flap. This flap should be made approximately 1/2 inch longer than the diameter of the leg at the level of amputation (Fig. 7). The deep fascia is cut at the same level of skin incision and is not dissected from the overlying skin. The anterior compartment of muscles are sectioned at the level of the intended tibial bone section. The fibula is exposed extraperiosteally, deliberately avoiding stripping the periosteum from the fibula, to prevent development of bone spurs. The fibula is easily transected with a Gigli saw, sectioning it only 1/2 inch proximal to the intended level of the tibial section.

Following the transection of the fibula, the tibia is approached and the area of bone section determined and marked by a sharp incision in the periosteum. The tibia is sawed through at this same level, always bevelling the anterior, distal end of the tibia to avoid a sharp bony point, which may subsequently erode through the stump skin. It is unnecessary to strip tibial periosteum proximally from the bone end, as is often recommended in older surgical textbooks. The posterior portion of the amputa-

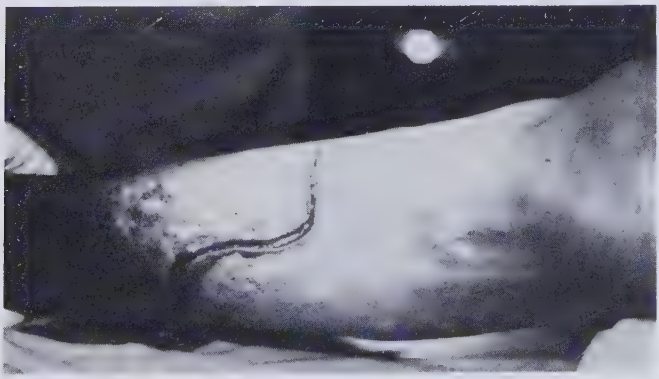

Figure 7. Long posterior skin flap formation.

tion is then completed, transecting muscle tissue at the level of bone section, being careful to identify and clamp the large posterior tibial vessels as one proceeds toward the posterior skin flap.

The muscles, particularly the gastrocnemius, can be transected down to the deep fascial flap and removed, or they may be scived down so that a thin layer of gastrocnemius muscle remains attached to the long posterior skin flap. If the gastrocnemius muscle has a good blood supply, and appears healthy, this technique may be the one of choice. If the gastrocnemius muscle appears gray, does not bleed, or shows evidence of necrosis, it should be resected to the appearance of normal muscle tissue. Major blood vessels are doubly ligated if there is any evidence of active bleeding, or ligated singly if they appear to have clotted. The major peripheral nerves (posterior tibial and peroneal) are identified and are dissected from the surrounding soft tissue with the finger. These major nerves are then gently stretched downward and sectioned cleanly with a scalpel as high as one can reach. The nerves in the below-knee area usually do not bleed sufficiently to require ligation. Neuromas will form at the ends of cut peripheral nerves, no matter how they are locally treated, so it is not recommended that the nerve ends be either ligated or injected with alcohol or phenol or any other noxious substance.

With complete removal of the ischemic lower extremity and with ligation of all bleeding points, the wound is then closed in two layers, the deep fascia being the first and the skin edges the second. Redundant skin is tailored to provide a snug but not tight skin closure (Fig. 8). No attempt is made to remove dogears, since these will quickly shrink and become no problem. Excessive soft tissue padding on the end of an amputation stump is to be avoided, because it may become edematous, which may be followed by stasis dermatitis and skin ulcerations.

Myodesis, or the practice of attaching muscle tissue to the stump end to provide a circumferential padding of the bone end, is not a well advised technique in peripheral vascular amputations. The muscle tissue at the amputation site usually has compromised blood flow, and to further embarrass its blood supply by mattress sutures only invites complications. If

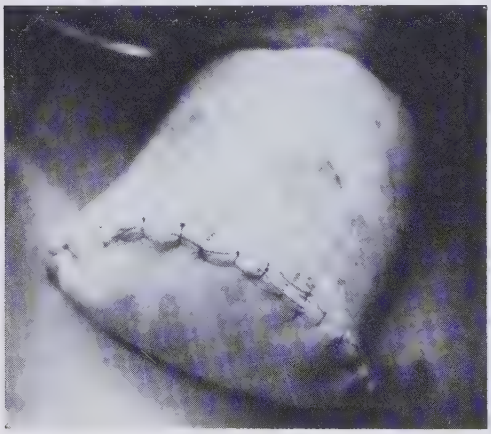

Figure 8. Skin closure with long posterior skin flap.

the muscle bellies have not been dissected from the surrounding deep fascia, closure of the deep fascia will pull the muscles down to provide circumferential padding as is provided by myodesis. Before the skin sutures are finally placed, a polyethylene tube drain connected to positive suction is placed in the subcutaneous or subfascial area. This tube avoids the bone end so as to avoid exsanguination of the patient due to exuberant suctioning of blood from the marrow. This drain is usually removed in 24 to 48 hours.

Immediate Rigid Plaster Dressing

Following closure of the stump end, it has been the practice of most orthopedic surgeons, particularly those trained in modern amputation techniques, to provide an immediate rigid plaster dressing. This consists of several 4×4 inch dressings over the wound itself, followed by a small amount of lamb's wool padding and the subsequent application of a Spandex stump sock. Padding of the bony prominences of the stump is then performed either by wrapping the stump with several layers of Webril sheet wadding, which eases the removal of the cast, or by applying a small felt pad over the patella and two long felt strips on either side of the tibial crest to flair laterally and medially over the tibial condyle edges (Fig. 9).[1] Subsequent to the padding, the stump is then covered by two rolls of elastic plaster, endeavoring to obtain a firm compression of the stump end, with the wrapping pressure being gradually reduced as one proceeds upward along the stump sock to above the knee (Fig. 10). The rigid plaster dressing can be molded above the medial femoral condyle to provide self-suspension of the rigid dressing (Fig. 11), or, if desired, a webbed belt can be incorporated in the plaster cast, which is then attached to a waist belt. Either method provides effective suspension of the plaster dressing while the patient is being ambulated.

At this point the surgeon makes a choice of sending the patient to the recovery room or going a step farther and applying a temporary pylon extension to the rigid plaster stump dressing. If a certified prosthetist is a member of the amputation team, he may apply the pylon attachment and SACH foot to the completed rigid stump dressing (Fig. 12).

When the patient awakes from his anesthetic, it can be with the visible evidence of a prosthetic foot attached to his rigid dressing, giving him a psychological lift, as this implies he may be ambulating soon. However, if a prosthetist is not available and the pylon is not applied, the patient may still be ambulated using crutches, parallel bars, or a walker within 2 to 3 days of the amputation, thus minimizing the debilitating effect of continued bed rest. The first rigid plaster dressing remains in place for 10 to 14 days. The sutures are removed at that time if the wound has healed satisfactorily.

Of course, if any complications develop during this first 2 week period, such as high fever, increasing stump pain, or an odor emanating from the cast, the rigid dressing is removed and the stump inspected. After inspecting the wound, a second rigid plaster cast is then immediately reapplied, utilizing the same principles of application as with the first cast. If the healing of the wound appears precarious, one can post-

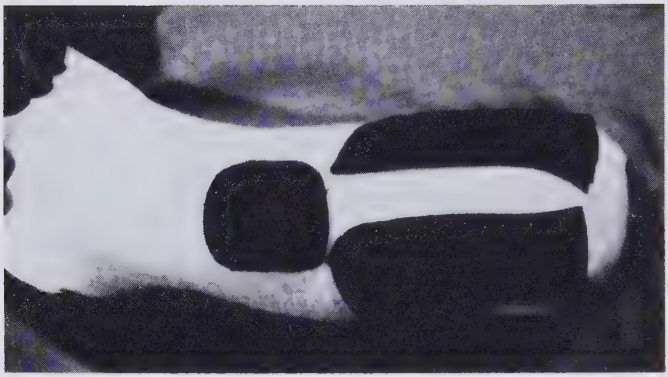

Figure 9. Wool-felt relief pads over patella and on either side of tibia crest.

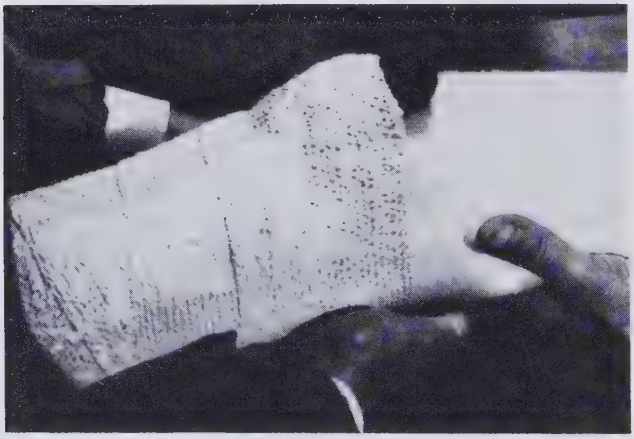

Figure 10. Application of elastic plaster wrap to stump.

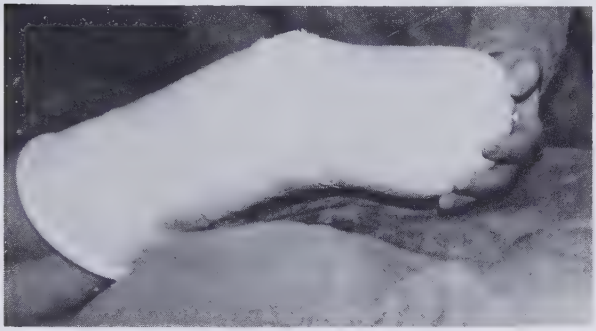

Figure 11. Padded rigid plaster stump dressing.

Figure 12. Pylon attached to temporary plaster socket for early ambulation.

pone the application of a pylon and subsequent weight bearing ambulation for a second period of 2 weeks. However, if adequate healing of the stump is present, and the tissues appear to be able to survive weight-bearing stress, then the patient can be seen in the cast room by the prosthetist, or can be sent to the prosthetic laboratory for the application of the proper length pylon and SACH foot.

The patient is then begun on an ambulation program under the supervision of a trained therapist. The patient is advised to place as much weight on his temporary prosthesis as is comfortable. The temporary stump cast is usually again changed 4 weeks postoperatively and at this time can be reapplied as a below-knee type socket, which will require pelvic belt suspension, but does allow motion at the knee joint. The previously used pylon is then reattached and the patient continues with ambulation. This third plaster dressing and pylon can be worn as long as the socket is comfortable and retains its fit. The patient is seen at subsequent intervals and the stump is inspected. When the physician is of the opinion that the stump is sufficiently mature, the patient is considered ready for his definitive prosthesis. The immediate rigid plaster stump dressing with or without pylon attachment has lent itself most admirably to the below-knee amputation and has become in most centers, a definite part of the routine treatment for the below-knee amputee.

The below-knee amputation patient, whether he be a young adult or a geriatric patient, is well served by a patellar tendon bearing prosthesis with condylar strap, or by one of the variations, either the supracondylar-suprapatellar type below-knee prosthesis, or the supracondylar variant of the patellar tendon bearing with removable wedge suspension.

THE ABOVE-KNEE AMPUTATION

As our ability to perform satisfactory below-knee amputations has improved, the above-knee level is no longer an automatic amputation level for peripheral vascular disease. However, there are occasional patients who may require an above-knee amputation because of the original extent of the necrotic process, and some below-knee amputations may have to be revised to the above-knee level because of further distal necrosis or breakdown at the below-knee level.[8] This complication is more frequently found in the patient who has arteriosclerosis obliterans without diabetes, or the patient who has a sudden onset of irreversible femoral occlusion, and who has not had sufficient time to develop collateral circulation. However, in most instances, even these patients are prepared preoperatively for either an above-knee or below-knee amputation. If adequate skin bleeding is present at the below-knee level, the amputation is completed at that level. In some instances, however, and particularly where femoral-popliteal bypasses or aortofemoral grafts have failed, the thigh amputation may be the procedure of choice.

The skin circulation above the knee in both the posterior and anterior areas is fairly symmetrical, and so skin flaps of equal length are incised with the expectation that healing of each will be satisfactory (Fig. 13).

Figure 13. Equal length skin flaps for above-knee amputation.

The incision is deepened through the fascia at the level to which the skin retracts. Muscles are sectioned approximately at the level of bone section, and the femur is transected without stripping of periosteum proximally. The sciatic nerve is usually ligated to minimize bleeding from the vas nervorum, which is nearly always present in this nerve. The major blood vessels are doubly ligated and all other bleeding points controlled.

No attempt is made to myodese the muscles surrounding the femur to the bone because of the probability of producing further muscle necrosis, which may delay stump healing. The wound is closed in two layers, the deep fascia first, then the skin. A polyethylene tube drain attached to a positive suction device, placed subfascially, aids in preventing subcutaneous hematomas.

The above-knee amputation may be covered with an immediate rigid plaster dressing modified to a quadrilateral brim shape by the use of the Seattle casting fixture (Fig. 14).[1] Suspension of the above-knee stump

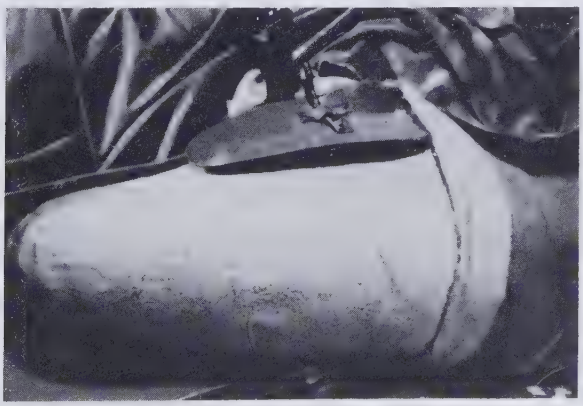

Figure 14. Application of above-knee rigid dressing using Seattle casting fixture.

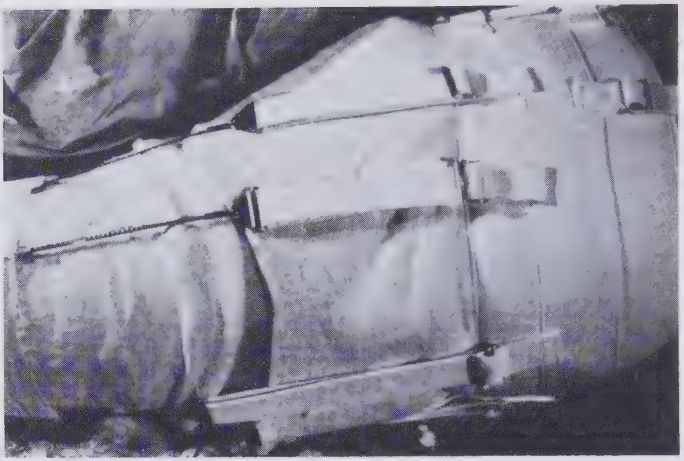

Figure 15. Above-knee suspension with felt waist corset.

cast is essential to prevent it from slipping down and producing edema of the distal wound, due to loss of stump contact. Suspension of this type of rigid stump dressing is achieved by control cables passed through housing applied to the medial and lateral sides of the stump cast. The ends of the cables are then fastened both front and back to a wool-felt waist belt to provide suspension of the socket (Fig. 15). In some instances, the use of the immediate rigid plaster socket for the above-knee amputee has not been practical, and we have continued to use the classic technique of wrapping the stump with compression bandages in the form of an elastic hip spica dressing, utilizing three 6-inch elastic bandages sewn or taped together. The bandages must go about the patient's waist to keep the dressing in place on the stump, and snugly applied into the adductor area in order to prevent the formation of an adductor roll (Fig. 16). In other instances, we have applied a hip spica plaster cast in order to maintain the rigid socket dressing in total contact with the healing stump. A great deal of attention and care has to be provided to the above-knee amputee in the postoperative period if a rigid immediate plaster stump dressing is to be successful. In all instances, however, once the sutures are removed, a rigid plaster socket is applied, utilizing the quadrilateral socket principles. This socket is then fitted with a pylon attachment with a knee articulation and a SACH foot. The patient, then begins ambulation on this temporary prosthesis, which has an articulated knee joint providing a similar function that his definitive above-knee prosthesis will soon provide.

Ambulation may thus be early, 2 or more days after the amputation itself; or it may be delayed, occurring 1 to 2 weeks after surgery. In either event, within a day or so the patient should be ambulating between parallel bars or with crutches to prevent the debility that comes from prolonged bed rest. If the patient's general condition is adequate, there is no excuse, prosthesis or no prosthesis, for keeping the patient in bed or in a wheelchair.

Figure 16. Classic compression spica elastic bandaging for above-knee amputee.

Rehabilitation following above-knee amputation is a great deal more difficult than for the below-knee amputation because usually the patient is fitted with a prosthetic knee articulation, which must be controlled during the stance phase. This control is achieved in part by "built-in" stability, which means that the knee center is aligned behind the weight bearing line, and by thigh and hip extension providing an extension moment at the knee, (when the foot is on the ground) providing a force to stabilize the knee in the stance phase. A Bock safety knee is an alternative prescription.

There are two methods of suspension for the definitive above-knee prosthesis; a pelvic belt, or positive air pressure suspension (suction socket). The limited ability and strength of the geriatric amputee is usually not sufficient for him to be fitted with a suction socket suspension mechanism. Accordingly, most geriatric patients are fitted with a pelvic belt suspension for the above-knee prosthesis. The socket shape of choice is always the quadrilateral shape.

In general, rehabilitation of the single above-knee amputee takes nearly twice as long as for the below-knee amputee. In most instances, independent ambulation is accomplished only by means of additional aids, such as a cane or single crutch. If the patient becomes a bilateral above-knee amputee, the chance of the geriatric individual ambulating with two articulated knee prostheses is very unlikely.[16] Most bilateral geriatric above-knee amputees are rehabilitated to wheelchair status with the use of prostheses only on rare occasions (clinic visits).

SUMMARY

Definitive amputation in the foot ischemia patient, secondary to arteriosclerosis with or without diabetes, should always be considered as a

very important first step in the process of rehabilitation. If the patient can have a below-knee amputation, the end product (effective, comfortable ambulation) is a great deal more satisfactory, than if the patient has an above-knee amputation. Only by preserving both knee joints can the surgeon anticipate adequate postamputation rehabilitation to an ambulatory status.

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Thoracic Outlet Syndromes

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> The bizarre symptoms and insidious onset render early diagnosis difficult, as does the fact that objective signs may appear relatively late.
>
> NAFFZIGER[12]

Differentiation in diagnosis of thoracic outlet syndromes has been dependent primarily on evaluation of highly subjective complaints and elucidation of imprecise physical findings. In addition, many tests utilized to confirm a diagnosis have been found to be positive in a high percentage of asymptomatic patients. In consequence, few surgeons have a clear view of these syndromes. Fewer still are confident of their ability to select patients for conservative or for surgical therapy.

Development of new techniques for quantitation of arterial and venous blood flow; selective, dynamic arteriography and venography; nerve conduction velocity studies and electromyography have been welcome. These present the physician with objective data to be utilized in making an early, precise diagnosis. Objective data thus obtained aid in selection of patients for physiotherapy or for outlet decompression. Serial studies help in measuring a patient's response to physical therapy or to surgery.

In the past, thoracic outlet compression syndromes were thought to produce symptoms primarily because of vascular impingement. In 1853, Hilton in England described subclavian artery compression secondary to a first rib exostosis in a patient who presented with gangrene of several fingers.[7] Later, Halstead and Holman reported post-stenotic aneurysmal dilatation of the subclavian artery in patients with a cervical rib.[6, 8] Because of early emphasis on arterial or venous occlusions, most diagnostic tests performed in the past during examination of a patient thought to have outlet compression relied on demonstration of vascular compression. Today, detection of significant neural compression utilizing nerve conduction velocity and electromyographic studies has emerged to become critically important in confirming a diagnosis of thoracic outlet compression. Nelson and Davis emphasize that neurologic symptoms are more frequent than vascular ones early in this condition.[13] Roos, in his

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series of more than 276 cases, pointed out that major vascular involvement was infrequent and that most of his patients' symptoms were related to neural compression across the outlet rather than to vascular pressure.[15]

DIAGNOSIS OF THORACIC OUTLET COMPRESSION

In order to establish the diagnosis of thoracic outlet neurovascular compression, a full understanding of the anatomy of the thoracic outlet and of the distal extremity is necessary. A comprehensive three dimensional view of the relationship of the subclavian vessels and the brachial plexus to the fixed musculoskeletal structures within the thoracic outlet is important. It is also necessary to be able to visualize the relationships of these structures during motion of the arm, shoulder, neck and thorax if one hopes to understand the signs and symptoms. Therefore, a preliminary review of the anatomy of the outlet is recommended before concentrating on diagnostic testing.[11]

History

The admonition to listen to the patient may be hackneyed and trite, but in diagnosing the thoracic outlet compression syndrome, there is no more important guide than a clear, concise history. The relationship of the patient's subjective symptoms to activity and posture are vital in making an accurate diagnosis. For example, radiation of pain over the C8-T1 dermatomes following abduction of the arm should suggest outlet disease, whereas pain limited to the median nerve distribution in the hand and fingers signifies entrapment within the carpal tunnel. Pain in the dermatomes of C4-C5 or C5-C6 is frequently related to herniation of a cervical nucleus pulposus. Diffuse neck and arm pain relieved by heat, exercise and aspirin suggests cervical arthritis or radiculitis. Clearly, examination of the patient must include a comprehensive neurologic examination to rule out primary disease of the central nervous system.

Special Maneuvers

The Adson test, the exaggerated military or costoclavicular maneuver, and the hyperabduction–external rotation (H.A.E.) test should be performed routinely. Attention should be paid to diminution of the radial pulse, development of a bruit over the subclavian or axillary arteries, or presence of peripheral venous hypertension. Production of pain in the neck, arm or hand which duplicates the patient's complaint is a most important diagnostic finding during these tests.

False positives are encountered in performing the special maneuvers. Thirty-eight per cent of the normal population will develop alterations of the radial pulse with the Adson test, 68 per cent with the costoclavicular maneuver, and 54 per cent with abduction of the arm. However, production of symptoms with postural changes is reliable and important.[18] The physical findings must then be correlated carefully with the patient's history.

Supraclavicular tenderness is a frequent finding in patients with the thoracic outlet compression syndrome. Palpation of a cervical rib, subclavian aneurysm, absent distal pulses, or ischemic changes in the hand, while uncommon, helps to confirm the diagnosis of outlet disease.

A positive Tinel's sign over the ulnar nerve at the elbow or the median nerve at the wrist should alert the examiner to look for entrapment at these sites. There is an unexplained coexistence of the carpal tunnel syndrome and the thoracic outlet syndrome. Perhaps edema of the hand and forearm secondary to the intermittent or persistent venous obstruction at the outlet produces constriction within the carpal tunnel. The most valuable single diagnostic test is the duplication of patient symptoms during one of the abduction maneuvers. Such symptom production need not be accompanied by pulse obliteration or induction of a supraclavicular bruit.

Conventional Roentgenograms

Routine x-ray studies should include chest, cervical spine and shoulder. The chest x-ray may reveal a superior sulcus tumor invading the brachial plexus, mimicking outlet compression. Cervical ribs, an elongated C7 transverse process, bifid first ribs, or malformed ribs or clavicles can also be seen on the chest and spine x-rays. Their presence helps to confirm the diagnosis, but again they are not diagnostic. A roentgenologic survey of patients with thoracic outlet compression syndrome seen at the Mayo Clinic revealed normal bony structures in 43 per cent, cervical ribs in 35 per cent, and abnormalities of the cervical vertebrae or upper rib cage in 22 per cent.[10] The presence of arthritic changes in the cervical spine or the shoulder may be of value in diagnosis. It should be noted that cervical ribs may be seen on 0.16 to 0.5 per cent of routine chest roentgenograms.[14]

Case Report. A 17 year old freestyle swimmer noted the spontaneous onset of severe upper chest and left shoulder pain while sitting in class in November 1972. Chest x-rays revealed a nondisplaced fracture of the left first rib. Of interest was a history of a fracture of the right first rib sustained 1½ years earlier. Over the next several months he complained of left shoulder and arm pain with radiation to the ring and little fingers. The pain and paresthesias were aggravated by elevation or abduction of the left shoulder.

Follow-up x-rays nine months later revealed nonunion of the fracture (Fig. 1). On examination he had mild left supraclavicular tenderness and positive Adson, costoclavicular and hyperabduction–external rotation tests on the left. Hyperabduction of the right arm to 110° obliterated the pulse in his asymptomatic side also. Significant neurovascular compression across the left thoracic outlet was confirmed by ulnar nerve conduction velocity studies.

Left transaxillary first rib resection produced prompt relief and has allowed him to resume normal activities, including swimming and weightlifting. During the operation the lower cord of the brachial plexus was found to be compressed by a large external callus at the fracture site (Figs. 2 and 3). There was marked hypertrophy of the anterior scalene muscle, suggesting that the fracture resulted from sudden contraction of the muscle.

Arteriography

Brachial angiography is of limited value and is not recommended. However, if vascular complications such as arterial occlusion, subclavian

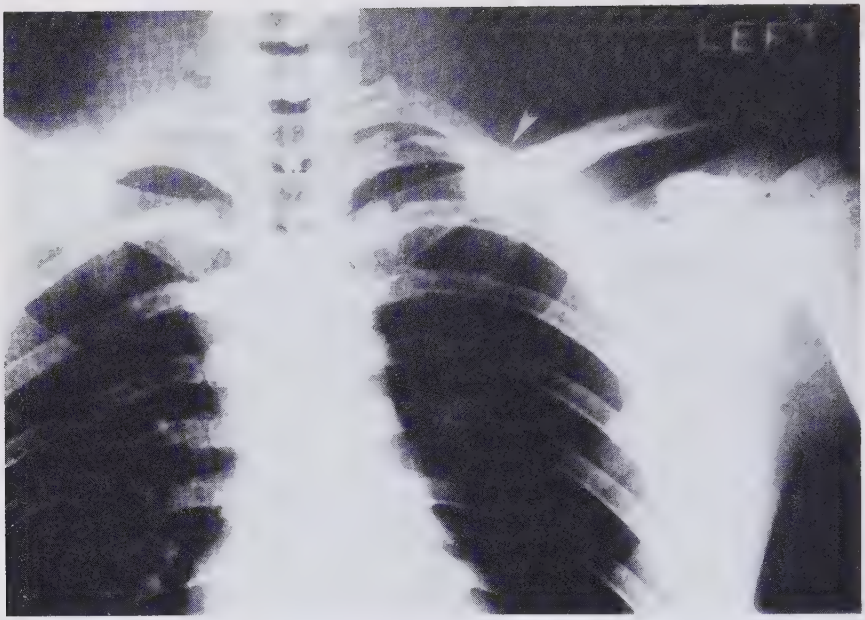

Figure 1. Non-union of the fracture of the posterior 1/3 of the first rib is seen on the routine chest x-ray. There is prominent external callus about the fracture site.

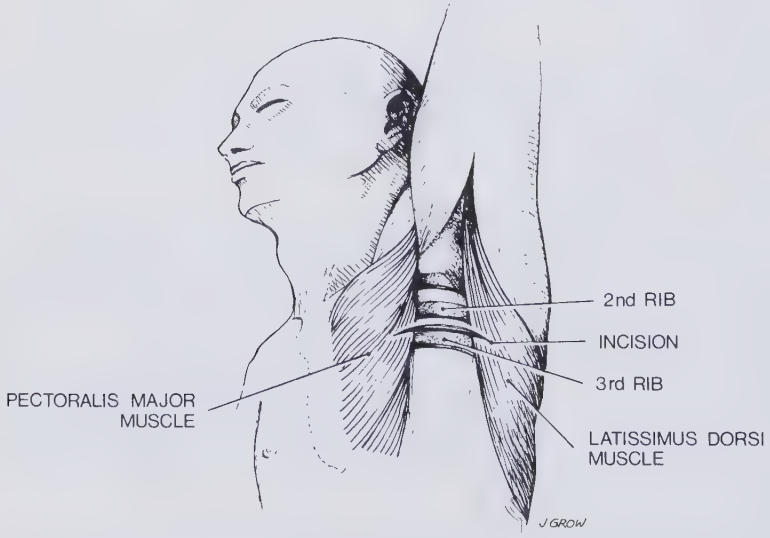

Figure 2. During surgery the patient is in a lateral position with the arm draped free. The transverse incision is just above the third rib and just below the hair line.

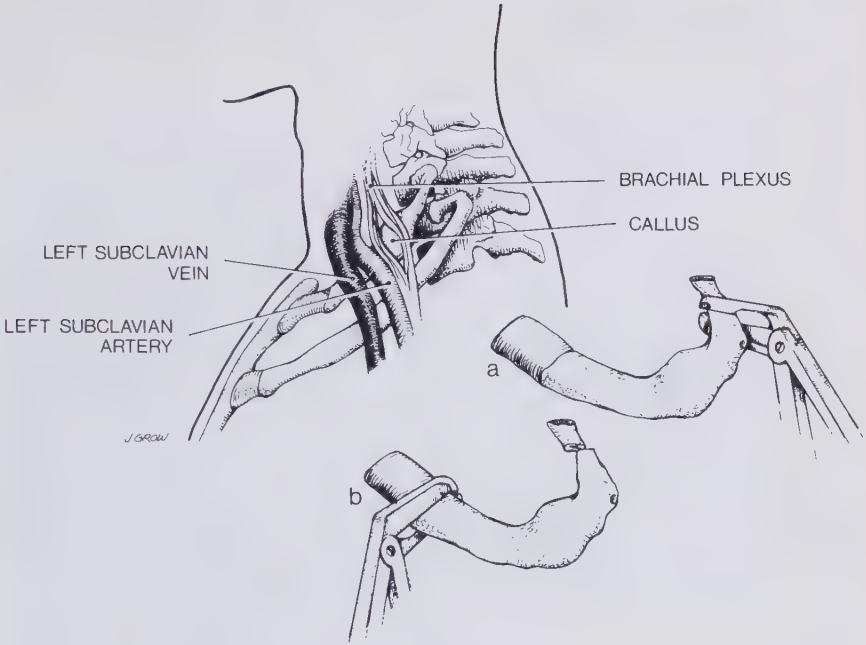

Figure 3. The brachial plexus is seen stretched over the callus. The anterior scalene muscle has been divided and the middle scalene dissected off the first rib. The insert shows the rib divided posterior to the plexus and anterior to the subclavian vein.

or axillary aneurysm, or embolic occlusion of distal vessels are suspected from the history or physical findings, dynamic subclavian angiography should be performed. Transfemoral selective subclavian angiograms should be performed with the arm in the neutral position and then repeated during postural maneuvers which produce alterations in the distal pulse. These may demonstrate the presence of arterial entrapment within the thoracic outlet.[2, 9] It should be emphasized, however, that arteriography is of very limited value and should be done only for specific indications as indicated above.

Venography

Functional venography carried out on the relaxed abducted arm and then with either hyperabduction and external rotation of the arm or with the shoulder braced backward and downward. Such venograms are indicated if there is a history of intermittent or persistent edema of the hand or arm or if a prominent venous pattern is present over the arm, shoulder or chest. Venous occlusion may be complete or intermittent. Obstruction of the subclavian or axillary vein causing prominent collateral veins with the arm in a neutral position suggests thrombosis. Venous occlusion demonstrated *only* with postural changes suggests intermittent subclavian obstruction across the outlet.[1, 9]

Resting peripheral venous pressure should be measured after venography is done. If the venous pressure is elevated or is normal and then

rises and remains high following exercise of the arm, major venous obstruction in the proximal upper extremity is present.

Ancillary Diagnostic Studies

Blood flow studies using the Doppler flow detector, pulse wave analysis, and impedance plethysmography have been done by us in a consecutive series of patients. These have been of limited clinical value in diagnosis of patients with the thoracic outlet compression syndrome. This has been disappointing but confirmatory of the fact that only 5 to 12 per cent of patients have major vascular occlusion.[5, 15, 17] However, in patients with arm or hand swelling, ultrasound studies have been an aid. Thrombosis of the axillary-subclavian venous segment is easily diagnosed by changes in the flow sounds heard over the cephalic, basilic and brachial veins and by changes in the flow velocity patterns. Impedance plethysmography may also be used to establish major venous occlusion.[22]

While arterial occlusions are rare in patients with thoracic outlet compression, detection of the occlusion is facilitated by ultrasound analysis. Large collateral arteries around the shoulder preserve distal pulses despite proximal arterial occlusion. Thus apparently normal pulses may be palpated distal to a subclavian artery occlusion. In these cases, it is to be expected that damping of the Doppler flow signal will occur. Furthermore, the flow pattern may change as the arm is manipulated through the various abduction maneuvers. The ultrasound techniques aid in separating patients with complete arterial occlusion from those with intermittent obstruction. Impedance plethysmography and ultrasound flow detection, being noninvasive, are ideal for performance of postural tests (Adson, costoclavicular and hyperabduction). These tend to confirm the diminution or cessation of arterial flow across the outlet but do not distinguish those patients whose symptoms are unrelated to incidental pressure upon the subclavian artery from those who have the thoracic outlet syndrome due to neural compression.[17, 18]

Nerve Testing

Since symptomatic neural compression frequently exists without concomitant major arterial compression, objective testing for nerve compression is important if reproducible information is to be obtained in patients with thoracic outlet compression syndrome. Nerve conduction velocity studies as defined and developed by Caldwell and then utilized by Urschel have added a new dimension of accuracy and objectivity in diagnosis of the thoracic outlet compression syndrome.[4, 20] In these tests, ulnar motor nerve conduction velocities are measured in the forearm and wrist, across the elbow and through the thoracic outlet. The average normal ulnar nerve conduction velocity across the thoracic outlet is 72 meters per second, 55 meters per second around the elbow and 59 meters per second in the forearm. Urschel found that patients with thoracic outlet compression syndrome had an average ulnar nerve conduction velocity reduced to 53 meters per second across the outlet with a range of 32

Table 1. *Normal Ulnar Nerve Conduction Velocity*

	JEBSEN	CALDWELL
Forearm	59.4 m/sec	59.1 m/sec
Elbow	55.7 m/sec	55.8 m/sec
Outlet	61.2 m/sec	72.2 m/sec

Modified from Caldwell.[4]

to 65 meters per second (Tables 1 and 2).[21] In his study, patients whose ulnar nerve conduction velocity was less than 60 meters per second rarely obtained relief of symptoms with conservative therapy.

Motor nerve conduction velocities are extremely helpful in differentiating thoracic outlet syndrome from degenerated or herniated cervical disks, radiculitis secondary to cervical arthritis, spinal cord disease, or pressure on the brachial plexus by tumor. In addition, these studies are helpful in diagnosing peripheral nerve entrapment in the carpal tunnel, at the elbow, or at the proximal humerus. They also aid in defining systemic neuropathies. When ulnar, median, radial and musculocutaneous nerves are evaluated in patients with outlet compression, the data obtained assist in making an accurate, objective diagnosis and are also useful as a guide for therapy.

From 50 to 70 per cent of patients with thoracic outlet compression will be improved by a course of physical therapy designed to improve posture and elevate the shoulder girdle.[16, 19] This is especially true of those who have relatively mild complaints such as fatigue, occasional numbness or tingling of the hand or arm. Shoulder girdle exercises may require 4 to 6 weeks before producing relief.[3, 5]

SURGICAL SELECTION

Patients with significant vascular symptoms and those with a cervical rib are those who have excellent relief of symptoms with surgical decompression of the outlet. Similarly, patients with obvious neurologic deficits or signs of major venous occlusion should have surgery recommended early. Utilizing the data obtained from nerve conduction velocity studies, Urschel found that the majority of patients who had an ulnar nerve conduction velocity about 60 meters per second improved with

Table 2. *Ulnar Nerve Conduction Velocity in Thoracic Outlet Syndrome*

	AVERAGE	RANGE
Normal	72 m/sec	68–75 m/sec
Preoperative	53 m/sec	32–65 m/sec
Postoperative	70 m/sec	58–85 m/sec

Modified from Urschel.[21]

physiotherapy. However, those who had conduction velocities below 60 meters per second required operative decompression of the outlet. Transaxillary first rib resection was shown to be the best method of accomplishing such decompression.[21]

SURGICAL THERAPY

Roos, Sanders, Dale and Urschel have all described excellent results with transaxillary first rib resection for decompression of the thoracic outlet. Ancillary resection of a cervical rib, abnormal fascial bands, division of the pectoralis minor tendon and resection of the second rib can be accomplished easily and rapidly utilizing the axillary approach. Therefore, this method is highly recommended as the procedure of choice in those patients requiring operative intervention (Figs. 2 and 3).[5, 15, 17, 19, 21]

Combination of precision of diagnosis by nerve conduction studies with completeness of outlet decompression by first rib resection has great theoretical appeal and should improve the results of therapy. In a series of patients evaluated and treated in this manner, Urschel reported 85 per cent good, 10 per cent fair and only 5 per cent poor results.[21] Complete relief of symptoms or improvement to a state of mild residual symptoms should be achieved in at least 90 to 95 per cent of all patients if all of the diagnostic tools are employed and adequate decompression is performed. Since poor results of treatment for thoracic outlet compression in the past were largely secondary to poor patient selection and in part to inadequate decompression of the thoracic outlet, objective analysis and total surgical ablation of the offending pressure point should solve the dilemma of the thoracic outlet syndrome.

SUMMARY

The thoracic outlet syndrome comprises a variety of symptom complexes occurring secondary to neurovascular compression within the thoracic outlet. The first rib usually acts as the primary pressure fulcrum in the development of symptoms. It then causes symptoms by compression of the subclavian artery, vein, brachial plexus, the C8-T1 trunk or various combinations of these structures. Neural symptoms include tingling, numbness or pain in the arm, hand, shoulder and neck, especially in the C8-T1 dermatomes. Clumsiness or weakness of the hand may occur in advanced cases. Venous occlusion may be intermittent or persistent and can cause edema, venous engorgement, cyanosis, a dull aching, heavy or bursting sensation which becomes worse following exercise, or none of these.

Diagnosis of thoracic outlet compression depends upon an accurate history accompanied by postural reproduction of symptoms with or without pulse change. Confirmation of the diagnosis requires routine roentgenologic survey of the thorax, shoulder girdle and cervical spine.

Motor nerve conduction velocity studies present objective reproducible diagnostic and prognostic data. They should be done routinely in every case. Dynamic arteriography, phlebography, arterial and venous flow studies aid in improving diagnostic accuracy in complicated cases, but are not to be considered routine.

Physiotherapy is the treatment of choice in the patient with minimal signs and symptoms, and operative therapy is reserved for patients with significant neurovascular signs and symptoms or those with persistent or recurrent symptoms following conservative treatment. Decompression of the thoracic outlet has been accomplished by scalenotomy, cervical or first rib resection, or claviclectomy. Of these, first rib resection produces consistent relief of symptoms with minimal morbidity and has become the preferred method of surgical treatment. Venous thrombectomy, arterial embolectomy or reconstruction are infrequently required. Neural compression is far more frequent than vascular compression in producing symptoms. Nerve conduction velocity studies and adequate decompression of the brachial plexus hold much promise in solving the problems of diagnosis and therapy of this condition.

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Surgical Considerations in Congenital Arteriovenous Fistula

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Congenital arteriovenous fistulae are manifest in a broad spectrum of lesions.[1, 3] Fortunately only a few require surgical intervention because of their systemic effect. Woollard described the developmental pattern of the vascular system[5] (Figs. 1 and 2). It can be appreciated from his drawings that arrest in development in the capillary or retiform stage would result in the common types of congenital arteriovenous fistulas. Arrest in development during the capillary stage gives rise to the port wine stain. This is of concern cosmetically but in itself has no systemic manifestation. It may be associated with other lesions such as the Sturge-Weber syndrome or large arteriovenous communications underlying the skin discoloration.

There is some confusion as to whether port wine stains are the precursors of large underlying arteriovenous communications. This stems from the fact that arrest in development is not always uniform and may be a combination involving the capillary network and larger vessels. Trauma probably plays little role in the development of these communications, but rather calls attention to a pre-existing lesion.

Arrest in development in the retiform stage results in large areas of communication between the arterial and venous systems. Since the communications are multiple there is extensive shunting of blood. The lesion may involve an entire extremity including bone or may be more limited. Unfortunately widespread involvement is common. The flow of blood through the inflow artery is excessive due to the low peripheral resistance. This results in dilation of these vessels. Whether it leads to eventual cystic medial wall degeneration as seen in long-standing acquired arteriovenous fistulas is not known. Certainly this is a well documented sequela in acquired lesions, as shown by Lindenauer et al.[2]

Large arteriovenous communications produce profound systemic manifestations which demand therapy. As these lesions progress, *three definite indications for surgical intervention become apparent:*

1. Heart failure caused by massive shunting of blood.

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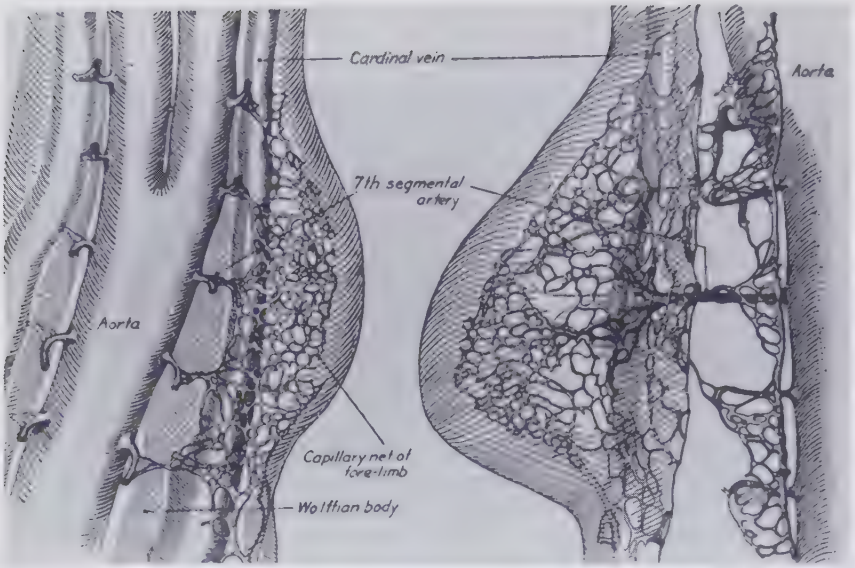

Figure 1. Embryologic development of the vascular system demonstrating the capillary stage. (Figs. 1 and 2 reproduced from Patten, B. M.: Human Embryology, 2nd ed. New York, McGraw-Hill Book Co., Inc., 1953, p. 633. Used with permission.)

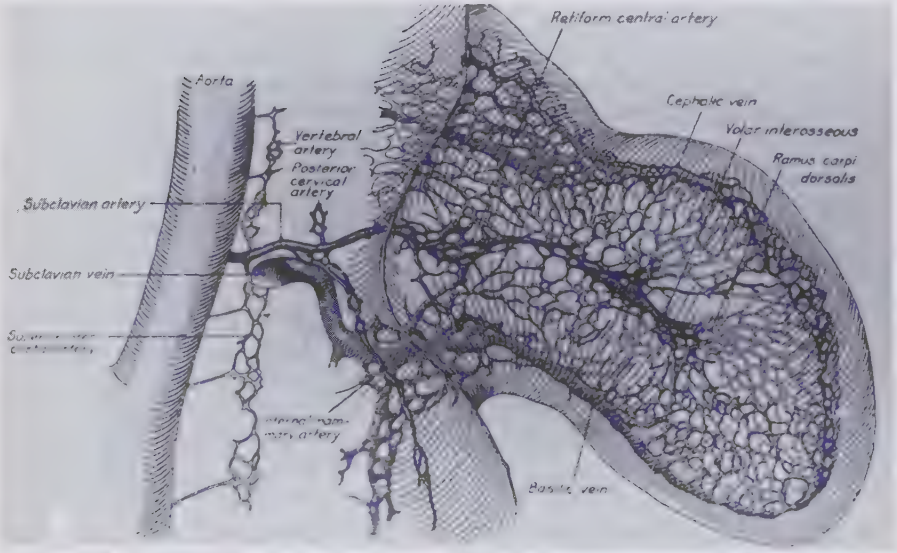

Figure 2. Embryologic development of the vascular system demonstrating the retiform stage.

2. Pain caused by large lesions encroaching on major nerves or weight-bearing surfaces.

3. Hemorrhage caused by a large lesion extending through the skin and becoming vulnerable to trauma.

Operative intervention is difficult and at times life-threatening. The existence of a lesion with none of the above indications does not constitute a reason for attempted operative extirpation.

The diagnosis of large congenital arteriovenous anomalies is usually made without difficulty on physical examination. The association of large dilated subcutaneous veins with a loud bruit is generally sufficient. There is usually a large spongy area of blood-filled vascular spaces which is easily palpable. One should carefully delineate cardiomegaly and signs of overt heart failure in any patient with a suspected congenital arteriovenous anomaly. One helpful maneuver is the use of the Branham sign as shown by slowing of the pulse with manual occlusion of the inflow artery. This is seen only with large lesions which have demonstrated cardiac decompensation. The difficulties encountered with the delineation of the extent of the lesion have largely been circumvented with the advent of careful selective angiography. The lesion can be accurately seen and classified by arteriography. The main feeding arteries can be demonstrated well. It must be remembered with the low peripheral resistance seen in congenital arteriovenous anomalies that the blood supply may have a bilateral origin. Therefore any arteriographic study must by necessity utilize a bilateral injection.

High venous oxygen saturation is seen if samples are obtained from the main venous drainage of the lesion. In addition there may be overgrowth of the part, particularly in extremity lesions.

SURGICAL PLANNING

Our therapeutic approach to this problem utilizes initial arteriography followed by operation with excision of as much of the lesion as possible. Following this, postoperative arteriography is utilized to demonstrate the aspects of the lesion still present, and usually a second operation is indicated in order to ligate all of the arterial inflow to the lesion and to effect complete extirpation of the arteriovenous anomaly. While it is true that these lesions tend to grow, in a particular case this may not happen. The initial visualization and surgical extirpations of these lesions attack only the central area where the large arteriovenous communications exist. Following extirpation of this portion, the smaller peripheral lesions tend to become more pronounced and one has the impression that the lesion is in fact a spreading neoplastic process. This is the reason that more than one operation is usually required to attempt complete extirpation of the lesion. As is shown in Case 2 many lesions are adequately handled by partial excision, as total excision would necessitate amputation of an extremity or complete irreversible disfigurement. The utilization of the combination of surgical and conservative measures has been very helpful in our series of cases.

The difficulties in therapy have been alluded to. However, an orderly plan for therapy must be emphasized. Case 3 demonstrates the staging procedures utilized in the care of a large arteriovenous anomaly of the face and scalp necessitating operation primarily because of bleeding, with a secondary indication being the disfigurement caused by the lesion.

If a patient meets the criteria for operation with either heart failure, hemorrhage or pain, one must utilize careful preoperative arteriography. It must be emphasized that many arteries will supply one of these anomalies because of the rich collateral network found in most areas of the body. Therefore, in the proximal extremities and in the head and neck, contralateral arteries must be visualized in order to accurately delineate their part in feeding arterial blood to these lesions. In addition, in lesions involving the face and scalp, one must visualize the intracranial vessels in an attempt to delineate any communications between the cerebral blood flow and the scalp or face as contributing arterial components.

Operation must then be accurately devised in an attempt to excise as much of the lesion as possible without creating major defects either in function or cosmesis. This will require the interruption of major arterial inflow as well as accurate ligature around the periphery of the lesion in order to interrupt the collateral flow into the area. This when combined with excision of the lesion will give one the best chance for a good functional result and on occasion a complete cure. All nerves must be preserved in order to maintain function. This is well shown in Case 3, where tedious dissection of the entire seventh nerve was necessary in order to preserve function of the facial musculature. The seventh nerve was traced from the stylomastoid foramen to the actual end points in the facial muscles. This was necessary in order to totally excise the arteriovenous communications over the entire side of the face. This type of careful dissection in the face of massive bleeding from the lesion requires a great deal of patience on the part of the surgeon. Following the removal of as much of the lesion as possible and ligation of the peripheral collateral arterial supply, the area must be covered. In the extremity there is usually enough skin available for primary closure. However, in the head and neck, it is often necessary to utilize a primary skin graft. This can be followed with a secondary procedure utilizing a pedicle graft to provide adequate coverage.

As is shown in Case 2 the involvement of the lesion may be such that surgical extirpation is not practical. In such cases the interruption of major channels of blood supply and excision of the central mass of the lesion will suffice if combined with conservative therapy utilizing a tight elastic support. This preserves function and eliminates the hazards of cardiac changes and pain.

The following are three case histories which demonstrate some of the problems encountered with congenital arteriovenous fistulas and the therapy utilized.

CASE REPORTS

CASE 1. A 32 year old physician gave a history of having a faint port wine stain over the left lateral aspect of the neck, extending up behind the ear. As-

sociated with this in the past six years had been an increasing number of "varicose veins." He discovered a bruit over the left parietal region approximately one week before being seen in the surgery clinic. Physical examination revealed a port wine stain over the left lateral portion of the neck with associated dilated subcutaneous veins and a loud, harsh bruit audible over the parietal and occipital area of the scalp. There was a spongy sensation to the neck and scalp region. There was no evidence of heart failure, nor was the lesion painful to palpation. Branham's sign was negative. Because of the recent increasing size of the lesion and the detection of an arteriovenous communication, an arteriogram was obtained (Fig. 3). This demonstrated a localized arteriovenous communication being fed by branches of the external carotid artery. Late films demonstrated fill of the large venous plexus in the mid cervical region.

The patient was operated upon and the external carotid artery was divided. The external carotid artery was dissected out so that as many of its branches were individually divided as possible. This was done to prevent any cross communicating circulation from the ipsilateral external carotid artery. Following this the area of the arteriovenous anomaly was excised along with as much of the subcutaneous enlarged venous plexus as possible. The port wine stain was not extirpated. Postoperatively the patient has done well with no recurrence of a bruit.

While this operation was palliative in nature it relieved the patient's prominent symptoms and has obtained a satisfactory result with a limited procedure.

CASE 2. A 4 year old child presented with overt heart failure. Physical examination revealed a thrill over the left femoral triangle and a spongy mass on the medial aspect of the thigh. The patient was treated with digitalis. An arteriogram revealed a large arteriovenous fistula within the thigh extending from the femoral triangle down to and including the upper portion of the calf. There was a large communication between the profunda femoris and the arteriovenous anomaly as well as multiple areas being fed by the superficial femoral artery (Fig. 4).

The patient was operated upon and the profunda femoris artery was ligated and the mass on the medial upper aspect of the thigh was excised as much as possible without causing dysfunction of the thigh musculature. The patient

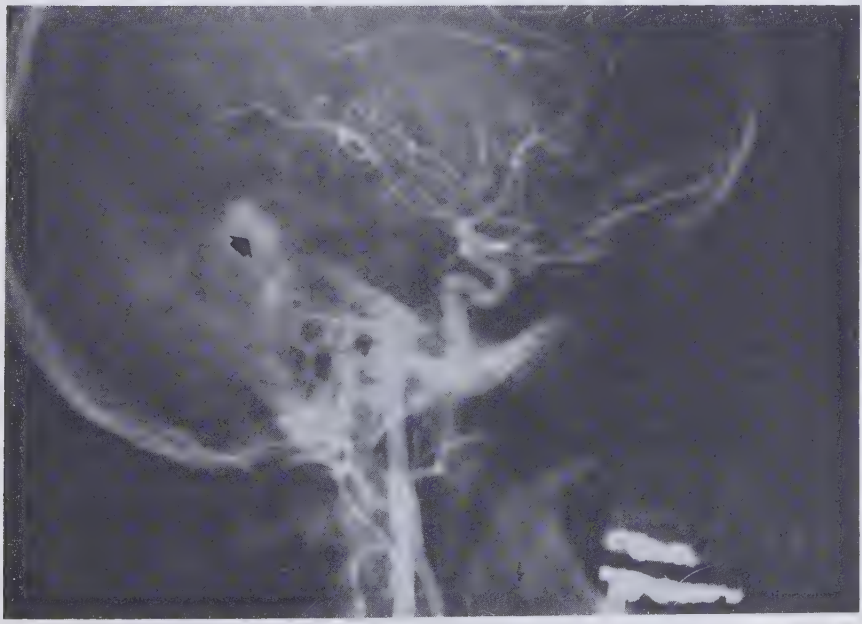

Figure 3. Case 1, demonstrating a small superficial congenital arteriovenous fistula being fed by the posterior-auricular artery. Notice normal internal carotid distribution.

Figure 4. Case 2, preoperative arteriogram (late phase) showing large arteriovenous anomaly.

recovered from this procedure without difficulty. Postoperatively there was no longer any evidence of cardiac decompensation. However, there was still an audible bruit in the lower thigh. The patient had a repeat arteriogram which revealed large communicating branches from the internal iliac artery on the left side feeding remnants of the arteriovenous fistula in the thigh (Fig. 5). The patient was reoperated upon through an abdominal incision and the entire left internal iliac artery was excised, including all of its branches. The patient tolerated this second procedure well and was fitted with an elastic Jobst leotard with a pressure of 40 mm Hg. In the subsequent 3 years the child has developed well without overgrowth of the left leg and without any signs of cardiac decompensation.

This case represents a partial excision of a large arteriovenous fistula to control the massive shunting of blood causing cardiac decompensation. Complete extirpation of this lesion would have involved a massive amputation as there was involvement of the distal femur and proximal tibia. There has been no subsequent skin breakdown and no other problems as a result of the arteriovenous fistula. The child has exhibited normal growth and development.

CASE 3. A 24 year old dental student presented after an episode of massive bleeding from the side of his face. Examination revealed a large arteriovenous anomaly over the mid portion of the cheek extending up onto the forehead and the temporal region of the scalp (Fig. 6). Arteriogram revealed a massive arteriovenous fistula being fed by the external carotid artery involving the cheek and scalp regions (Fig. 7). He was operated upon, at which time the external carotid artery was ligated and divided and the multiple branches supplying the face were dissected up into a large arteriovenous mass. A long dissection ensued with a total parotidectomy with preservation of the seventh nerve. Following this a large mass of skin overlying the lesion along with scalp was excised, with all of the arteriovenous communications being extirpated. Immediately the patient's skin defect was covered with a split-thickness skin graft. At a later time a pedicle graft was fashioned to cover the area with hair (Fig. 8). The patient remains totally

Figure 5. Case 2, arteriogram after first operation showing a small lesion after subtotal excision. Note absence of profunda femoris artery. Larger left internal iliac artery was excised at the second operation.

Figure 6. Case 3, appearance preoperatively showing large lesion.

Figure 7. Case 3, preoperative selective external carotid arteriogram showing massive arteriovenous anomaly.

Figure 8. Case 3, postoperative appearance of patient after total reconstruction.

symptom-free after 5 years with an apparent cure of a very large arteriovenous anomaly.

The indication for operation in this case was hemorrhage and severe cosmetic deformity. The operation was staged to allow adequate excision of the lesion along with adequate cosmetic reconstructive procedures. The patient was fortunate in that none of the lesion involved bone or any of the other vital structures in the face that may have precluded its excision.

New techniques utilized to treat congenital arteriovenous anomalies are few. However, a recent report by Taren et al.[4] indicates that intravascular coagulation may be helpful in controlling some aspects of these lesions. Because the entire lesion cannot be coagulated by this technique, prompt recurrence is the usual finding. This type of investigative effort, however, seems to bear fruit in that perhaps better techniques will be developed that can eventually obliterate these lesions by selective catheterization of multiple arteries with electrocautery.

In summary the arteriovenous anomaly poses a difficult problem to the surgeon. These lesions should generally be treated conservatively as wide surgical extirpations generally result in deformity and significant loss in function. Absolute criteria for operative procedures must be adhered to and when these are combined with the use of elastic support the results, while not perfect, are satisfactory for function.

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Modern Management of Abdominal Aortic Aneurysms

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> . . . but the really great advance and the introduction of a genuinely new idea was that of Voorhees, Jaretzki, and Blakemore. They introduced the entirely new concept of replacing a segment of the arterial system by a tube of partially porous and pliable plastic cloth.
>
> C. G. ROB, 1972[27]

Many surgeons reading this volume may recall being taught that abdominal aortic aneurysms were a natural consequence of aging. That attitude of uninformed complacency of the 1940's was replaced by one of frightened concern due largely to a milestone report by Estes from the Mayo Clinic.[15] His paper, appearing in 1950, showed that aneurysm disease of the abdominal aorta was more quickly lethal than untreated carcinoma. Advances were swift. Within a year, Dubost reported successful excision and graft replacement of an aortic aneurysm. Thus, all prior therapy became obsolete. This had included cellophane or cutis graft wrapping of the aneurysm, injections of diacetyl phosphate, or wiring with exorbitant lengths of intrasaccular wire with and without application of electric current.

With a single operation, Dubost moved aneurysm surgery away from indirect therapy characteristic of all methods prior to 1950, to the direct restorative surgery which all vascular surgery became in the latter half of the twentieth century.

By 1961, aortic surgery had advanced and aneurysm resection was included in a *Surgical Clinics* volume dedicated to standard operations. Since then, the natural histories of treated and untreated aneurysms have been defined, techniques of repair have been simplified markedly, and unusual associated conditions have been described, so that almost no circumstance need be a surprise to the aneurysm surgeon.

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INCIDENCE AND ETIOLOGY

The aortic aneurysm usually appears in an elderly male. Only 15 per cent of all patients with this condition are women and in most large series of cases, the mean age of patients is over 65. While the exact incidence of this lesion has not been determined definitively, the following reports attest to its frequency. When abdominal roentgenograms were used to screen 1400 ambulatory working subjects, 4.7 per cent had dilatation or aneurysm of the abdominal aorta.[29] Cranley found that the incidence varied with the year in which published series were collected, the population under study and the institution reporting.[9] His collection of incidences varied from 0.7 to 3.3 per cent. Finally, a careful study from Sweden indicates an incidence of 1.8 per cent of 5000 postmortem examinations. Thus, abdominal aortic aneurysms are common. At least 10 to 40 persons per thousand population over age 50 harbor these lesions.

It is said that arterial degeneration is the most common cause of aortic aneurysm. This is an explanation of etiology which is unacceptable, lacks precision, and fails to explain the reasons for the usual site of predeliction just distal to the renal arteries. An attractive theory of etiology was proposed by Benjamin in 1967. This was that occlusion of vessels nutritive to the aortic wall caused the degeneration in tensile strength which led to aneurysm formation. He showed that vasa vasora in the abdominal aorta were fewer in number than in the thoracic area and failed to cross through the vascular wall from one side to the other. Since vasa vasora are first branches of lumbar arteries and these are frequently occluded in patients with aortic atherosclerosis, it is to be expected that blockage of the aortic wall blood supply causes necrosis and subsequent healing by inelastic scar. Such a process explains the well described histologic features of transverse fractures of elastic lamellae, reactive collagen formation with depletion of smooth muscle cells, and, later, calcium salt deposition, the last phase of healing of the degenerated arterial wall.

DIAGNOSIS

Unlike other lesions of atherosclerosis, the abdominal aneurysm does not usually cause chronic symptoms. Neither does it often cause acute symptomatic occlusion. Instead, it most commonly produces a single catastrophic calamity: rupture. Since symptoms are lacking, the diagnosis of this lesion depends upon physical findings and roentgen confirmation. The finding of an expansile, pulsatile upper abdominal mass is diagnostic. Both lateral walls of the aorta must be identified, however, and the examining hands placed against these walls must move apart during pulsation. If the technique is observed carefully, even aortic aneurysms of 3.5 to 4 cm may be defined accurately.

While it has been emphasized that aortic aneurysms are symptomless and rupture of the aortic wall is calamitous, exceptions do occur. Chronic, severe low back pain may be present in association with an aortic an-

eurysm. Such pain is sometimes relieved by aneurysm resection. Similarly, an occasional patient will experience leakage of blood from the aneurysm weeks to months prior to diagnosis. This causes a reactive fibroplasia manifested as severe, dense, often thick perianeurysmal retroperitoneal fibrosis. This must be regarded a fortunate exception to the rule that disruption of the aortic wall is rapidly fatal.

Roentgenograms of the abdomen exposed in the anteroposterior and particularly in the lateral plane are diagnostic of abdominal aneurysms in 55 to 85 per cent of cases.[26] This supplements the 88 per cent accuracy of physical examination in evaluation of this condition.[15] In selected patients, obesity, scoliosis or pancreatic cysts may create diagnostic difficulty. Similarly, retroperitoneal lymphoma or sarcoma, or rarely lymphatic cyst may transmit aortic pulsations with sufficient force to cause diagnostic difficulty. In these patients, reflected ultrasound or isotope aortography may be of value. In the ultrasound method, a laminograph of compound-beta scans is used. The combination transducer-receiver emits 200 one-microsecond pulsations of 2.0 megaHertz/second, and portrays reflections on a storage oscilloscope from which Polaroid photographs can be made. The abdomen is scanned transversely and longitudinally from 4 cm. below the iliac crest upward until lung markings obscure the study.

Radionuclide visualization of aneurysm disease has a high potential for future use. Its place in diagnosis or in screening of patients is as yet undefined. However, its simplicity of performance, its nearly noninvasive character and high degree of patient acceptability prompt description at this time.

In performing radionuclide aortography, the patient is placed in supine position on the standard cart. He is then positioned with an Anger type scintillation camera centered over the suspected aneurysm. Ten to 15 millicuries of technetium-99m labeled serum albumin or technetium-99m as the pertechnetate is injected into a medial antecubital vein. At the time of injection, data recording is begun on a videotape system and images are taken. Approximately 1 million counts are collected on tape during a 60 to 90 second interval. This concludes the patient's portion of the test.

The tape is then played back and the optimal time for visualization of the aneurysm noted on a persistence oscilloscope. Images are then taken off the tape onto 70 mm or 35 mm Polaroid film using a motorized camera filming at 2 seconds per exposure. For evaluation of the renal arteries above the aneurysm, the technique is almost identical, except that the camera head is placed posteriorly rather than anteriorly.

The camera is fitted with a high sensitivity collimator. The volume of injection is between 1 and 5 ml, preferably 1 to 2 ml, and an 18 to 21 gauge needle is utilized. The tourniquet is kept above the site of injection until injection is completed, and then is released suddenly.

In 30 patients studied by the above technique in this institution, two false negative studies occurred. Both were in patients with large aneurysms and with thick laminated luminal clot.

Aortography has been abandoned by most for routine diagnosis of ab-

dominal aortic aneurysms, but it is employed as an aid in delineating involvement of the renal arteries. While Smith advocates translumbar aortography,[33] others have found catheter aortography both safe and rewarding.[10, 21] Robiscek found that, in 283 patients with aortic aneurysm, 29 had extension of the lesion into or above the renal arteries.[28] An additional six patients were found to have a more proximal aortic aneurysm. Performance of aortography aided these workers in deciding against aortic surgery in eight of the more aged patients. There was an initial error of 21 per cent in interpreting these aortograms. The aneurysm was not visualized in 48 of 218 patients.

UNTREATED ABDOMINAL ANEURYSM AND SURVIVAL

It has been difficult to ascertain the natural history of an untreated abdominal aortic aneurysm. Retrospective reviews are unsatisfactory, and selected lists of nonoperated patients are weighted with aged patients with secondary disease and those patients with small early lesions. Necropsy surveys contain errors in historical documentation (Table 1). Despite these objections, there are several important series, among which is that of Estes, who reported the fate of 102 patients seen at the Mayo Clinic up to 1947. This is generally thought to be a group of patients with abdominal aortic aneurysm seen before the era of surgical resection. Seven patients of 37 observed for 5 years survived, and this figure of 18.9 per cent has been taken to be the 5 year survival of patients with nonoperated abdominal aortic aneurysm. It is acknowledged that many patients seen in this group were those with early aneurysms or those with large expanding aneurysms referred to the clinic because of symptoms.

Later, Sommerville, Allen and Edward collected 178 autopsied cases of abdominal aortic aneurysms at the Mayo Clinic between 1925 and 1954. The survival time in this group of patients was available for 38 patients, three of whom lived for 5 or more years. The authors compared their data with that of Estes' previous report and concluded that the average earlier time of death after diagnosis in this later series reflected inherent selection of patients in an autopsy study.

Schatz et al.[29] reviewed the records of an additional 141 Mayo Clinic

Table 1. *Survival of Patients with Unresected Aortic Aneurysms*

AUTHOR	NO. PATIENTS	ANEURYSM RUPTURE	YEARS SURVIVAL 1	2	3	4	5
Schatz et al.*	119	44%	87.6%	–	52.5%	–	36.4%
Wolffe and Colcher	33	–	–	–	–	–	30.0%
Klippel and Butcher	30	10%	–	–	–	–	30.0%
Szilagyi	163	35%	–	–	–	–	17.2%
Estes	102	63%	–	–	–	–	–
Foster et al.	57	33%	63.0%	–	–	–	18.0%
Bernstein et al.	26	–	–	–	–	–	7.6%

*All patients followed less than one year excluded.

patients with abdominal aortic aneurysms first seen between 1950 and 1960, early in the period of surgical resection. Each of these patients had either elected to defer excision of the aneurysm (27 patients) or was not offered an operation (114 patients). Of the latter, 96 were not offered surgery because of extensive cardiovascular disease, six because they were over 80 years of age, and 12 because the aneurysms were very small. Despite the apparently poor outlook in the majority of these cases, 33 patients were followed for a full 5 years or until death with 12 survivors (36.4 per cent).

Wolffe and Colcher[38] presented an oft-quoted study in which a 5 year survival rate of at least 30 per cent for 33 untreated patients was documented. In another careful modern clinical study of 30 surgically untreated aneurysms, Klippel and Butcher[22] found a 30 per cent 5 year survival rate in a series which included a number of patients considered unfit for surgery. Szilagyi et al.[35] presented a 5 year survival rate of 17.2 per cent for 163 untreated patients. This group was also heavily weighted with surgical rejects. Of 223 patients evaluated, 96 were considered unsatisfactory surgical risks and, in 27 others, surgery was considered inadvisable by the patient's physician. In both this group and Klippel and Butcher's, there was a large number of patients with small aneurysms and advanced age who were considered safe risks without surgery.

All of these reports and extensive personal experience among experienced vascular surgeons has led to the generalization that any patient with an aortic aneurysm has three times greater chance of dying of aneurysm rupture than of surgical resection.

FACTORS INFLUENCING SURGICAL DECISIONS

Ultimately, there are three factors which influence the decision toward surgery in patients with abdominal aortic aneurysms. These are the patient's age, the size of the aneurysm and associated medical conditions. These facts are weighed against the background of knowledge that abdominal aortic aneurysms are potentially lethal and their behavior is totally unpredictable.

Age itself is not normally a contraindication to abdominal aortic aneurysm resection, but becomes a relative consideration at the extremes of age encountered in these patients. At ages over 70, associated medical conditions become increasingly important. Among these, pulmonary insufficiency is the most troublesome, renal insufficiency the most overemphasized, and senility the most philosophically important. When these or myocardial ischemia are present, a balance must be struck between dangers of surgery and risk of rupture of the aneurysm.

Some absolute contraindications to aneurysm resection include: acute myocardial infarctions, intractable congestive heart failure, intractable angina pectoris, severe pulmonary insufficiency, chronic renal insufficiency with a blood urea nitrogen higher than 80 mg per 100 ml, and associated fatal disease with less than 2 years' life expectancy.

Regarding aneurysm size, the figure of 6 cm transverse diameter has

become an accepted dividing line in aiding decision for or against resection. The aneurysm is measured on the abdominal physical examination and on a straight lateral film of the abdomen, which shows the aneurysm anterior to the lumbar spine. Aneurysms smaller than 6 cm in patients under 65 years of age are resected, but those in patients above 65 years of age and particularly those in patients with associated conditions as indicated above are usually observed. Abdominal films and physical examination are repeated at 3 month intervals. If the aneurysm shows increase in size, it is resected.

From Szilagyi's data,[35] the following conclusions can be obtained: A patient with an aneurysm 6 cm or less in diameter has a 75 per cent probability of surviving 1 year from the time of diagnosis, and a 47.8 per cent probability of survival for 5 years. A patient with an aneurysm greater than 6 cm in diameter has only a 50 per cent chance of surviving 1 year, and has a 43 per cent probability that this aneurysm will rupture. His chance of surviving 2 years is 25 per cent, and only 6 per cent that he will survive 5 years.

A summary of several reports on hospital mortality and long-term survival in patients who were treated surgically is given in Table 2.

ELECTIVE ANEURYSM RESECTION

The patient to undergo elective aneurysm resection is admitted to the hospital several days in advance of his operation. During preoperative preparation, pulmonary ventilatory function is assessed and pulmonary therapy given if needed. All smoking is prohibited and ancillary laboratory studies of hepatic and renal function are obtained, as well as an electrocardiogram and chest film. Aortography is performed only when hypertension is present, or if the aneurysm is thought to involve the renal arteries. Intravenous pyelography is desirable.

There is nothing to be gained from preoperative prophylactic digitalization nor from bowel preparation with a nonabsorbable antibacterial agent. Neither is mannitol used in preoperative preparation of the patient for surgery. An overnight infusion of 1000 ml of noncolloid fluid is given to insure preoperative hydration. An antibiotic effective against staphylococci and E. coli is started 24 hours prior to surgery and continued 10 days postoperatively. After induction of anesthesia, a central venous catheter is placed, as is a catheter in the urinary bladder. In patients of advanced age or with large abdominal aneurysms, an arterial pressure and blood gas monitoring line is used.

Absolute sterility is mandatory in operations in which a prosthetic graft is to be placed, and in order to assure decreased chance of contamination, a reasonable degree of surgical speed is also desirable. Although the abdominal aortic aneurysm can be resected through a transverse incision or through an extraperitoneal approach, both of these methods render an otherwise simple operation needlessly complex. Our own preference is for a vertical incision which can be closed carefully in several layers. A modern trend toward increasing use of the midline incision sup-

Table 2. *Mortality and Long-Term Survival of Patients with Aortic Aneurysm Resection*

AUTHOR	INSTITUTION	YEAR	NO. OF PATIENTS	HOSPITAL MORTALITY	YEARS SURVIVAL				
					1	2	3	4	5
Foster[16]	Vanderbilt	1969	116	–	70%	60%	–	–	50%
Gardner[17]	W. Va. Univ.	1970	56	5.3%	–	–	–	–	–
Baker[2]	Pennsylvania	1970	390	6.6%	–	–	–	–	–
Baker[1]	Pennsylvania	1970	240	9.5%	82%	76%	68%	61%	54%
Sanger[28]	Charlotte Mem.	1968	150	6.6%	–	–	–	–	–
Levy[24]	Barnes	1965	100	17.0%	76%	72%	–	–	34%
Voorhees[37]	Columbia	1963	170	18.0%	–	–	–	–	52%
May[25]	Rochester	1968	135	13.0%	–	–	–	–	49%
Hardin[19]	Kansas	1964	109	8.0%	–	–	–	–	–
DeBakey[12]	Baylor	1964	1449	9.0%	84%	–	–	–	58%
Vasko[36]	Hopkins	1963	136	9.6%	–	–	–	–	30%

ports its use. Once the peritoneal cavity is opened from the xiphoid to the pubis, a total abdominal exploration is done, attention being turned at last to the retroperitoneum. Thus, unanticipated lesions can be found prior to resection of the aneurysm and appropriate judgment exercised regarding their treatment.

The transverse colon is reflected upward and the small bowel to the right and upward. This is packed away with wet towels and large abdominal retractors are placed. The retroperitoneum is opened vertically in the midline, carefully avoiding the inferior mesenteric vein. The incision divides the ligament of Treitz and allows reflection of the duodenum to the right side. The renal vein is visualized and the infrarenal aorta is dissected free of investing areolar tissue. This first step in evaluating the aneurysm allows proximal control of the aorta below the renal arteries. If the aorta is not encased by surrounding inflammatory tissue, it is encircled with atraumatic Silastic tubing. When a marked inflammatory reaction is present, it is not necessary to isolate the posterior wall of the aorta. Side-to-side clamping is utilized instead of anteroposterior clamping. When a very marked inflammatory reaction is found around the aneurysm itself, this reaction is almost never above the renal arteries. However, it is necessary to dissect the renal vein free from the inflammatory mass so that the relatively normal aorta can be found inferior to the vein. If the aorta is markedly dilated adjacent to the renal arteries, as in severely hypertensive patients, it should be remembered that this type of aorta takes suture very poorly and has a tendency to tear during graft placement.

Following isolation of the aorta above the aneurysm, attention can be turned to distal control. If the common iliac arteries are not aneurysmal, they need merely to be stripped of investing areolar tissue on the anterior and lateral surfaces so that clamps can be applied. It is not necessary to completely encircle these arteries. If, on the other hand, dilation of the common iliac arteries is present, attention is directed to the external iliac artery, either distal to the colon on the left side, or in the pelvis on the right. Usually, the artery will be suitable for anastomosis and will be free of aneurysmal or occlusive disease. When proximal and distal control has been achieved, the patient is given 5000 units of heparin. Following this, proximal and distal clamps are placed and the aneurysm entered. It is evacuated of its laminated clot and open lumbar arteries are suture ligated from within. If the inferior mesenteric artery back-bleeds into the aneurysmal sac, it is ligated. Such back-bleeding insures that the colonic blood supply is intact. No attempt is made to endarterectomize the aorta since this causes needless bleeding. The overall technique is similar to that shown in Figures 1 through 5.

The graft is then sewn in place without transecting the aorta, utilizing a continuous, over-and-over 3–0 Dacron suture. Frequent backstitchings are advised so that there will be minimal leakage from needle holes. In patients with juxtarenal aortic dilatation in which the aortic wall is thin, it is possible to create a cuff of aortic wall which can be doubled onto itself to take suture better. Two thicknesses of aortic wall can then be used for the suture line rather than a single thickness. This

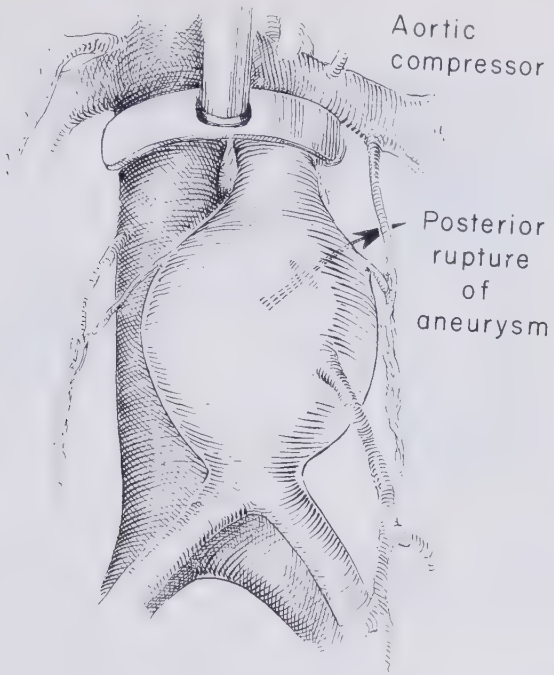

Figure 1. The aortic compressor may be placed as illustrated here, above the aneurysmal mass, or in the lesser sac, well away from the periaortic hematoma. In the elective aneurysmectomy, the compressor is not necessary.

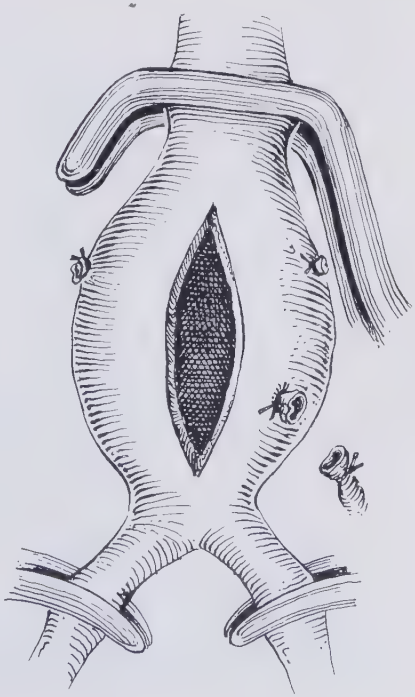

Figure 2. Linear aortotomy follows adequate proximal and distal control of the arterial stream. This control can be accomplished as shown or in other ways, depending upon associated aneurysms and arterial occlusions.

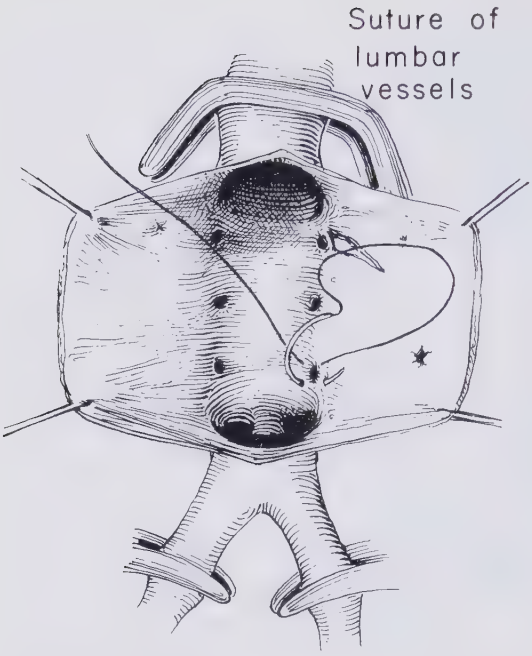

Figure 3. Each lumbar artery which back-bleeds is suture ligated from within the aneurysmal sac prior to graft placement.

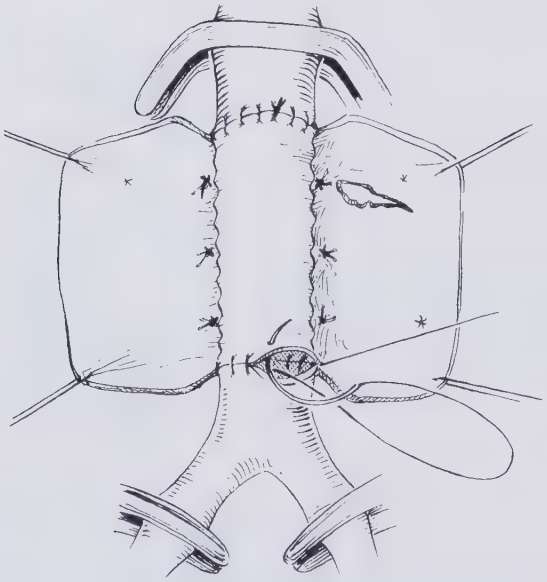

Figure 4. There is no need to transect the proximal and distal aorta in order to anastomose the graft. Simplicity is the key to successful arterial surgery.

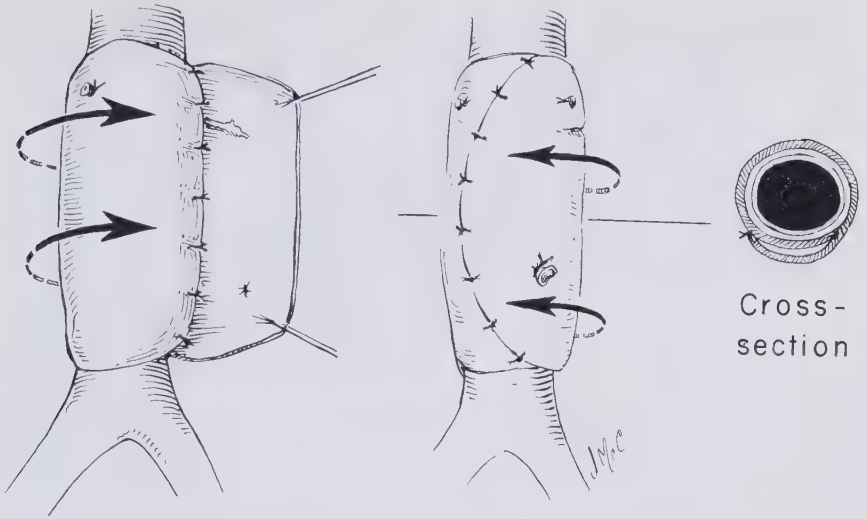

Figure 5. The aortic wall is not endarterectomized but is evacuated of contained clot. It is wrapped around the graft in order to cover the suture lines and protect the bowel from them.

minimizes aortic tearing, and helps to tamponade needle hole bleeding. The proximal suture line is tested before attention is turned to the distal suture line.

If the distal suture line itself is to be in the aorta above the iliac bifurcation, it can be placed without transecting the aorta. Thus, dissection damage to the vena cava or underlying iliac vein can be avoided.

Aortic surgery is similar to massive trauma in that interstitial fluid sequestration occurs. This must be replaced by intravenous fluid, and since aortic resection is so simplified, rapid fluid replacement has become an anesthetist's problem. The surgeon must be aware of this and should insure that three-fourths of the intravenous fluid and colloid replacement to be given during the surgical procedure is infused during the placement of the graft. We give 3 liters of noncolloidal fluid before aortic declamping. If this precaution is not taken, a period of hypotension may lead to postoperative renal failure. Fogarty catheters are used in every case to clear the distal circulation of any possibility of clot which may have formed during dissection or aortic clamping.

Clamps are removed first to one hypogastric artery, then to the lower extremity, and then to the other side in the same order. Blood pressure stabilization is achieved before each declamping.

RESECTION IN EMERGENCY CIRCUMSTANCES

Immediately after a patient is seen with a rupturing or expanding abdominal aortic aneurysm, the nearest physician should notify the vascular surgeon and move the patient to the operating room. A prominent no-

tice should be placed in each hospital emergency room to the effect that "The place to work up a patient with a rupturing aneurysm is in the operating room." Preparations in the surgical suite include placing two large intravenous catheters and a bladder catheter. While this is being done, blood is drawn for typing and cross-matching as well as preliminary baseline laboratory testing. The patient's abdomen is prepped and draped, and no anesthesia is given until the surgical team has assembled and instruments are available for the operation. If possible, the operation is not started until blood is available in the operating room for transfusion.

The surgical team must be ready to start the operation immediately upon induction of anesthesia, since it is at this time that a patient will frequently lapse into deep shock. As soon as the endotracheal tube is in place, the abdomen is opened rapidly, with care being taken to avoid bowel injury.

Unfortunately, nearly one-half of patients with ruptured abdominal aortic aneurysms arrive in the operating room in less than the ideal circumstances indicated above. In these patients, the abdomen must be prepped and draped rapidly, and the operation begun even before anesthesia is started. It is in these patients that the abdominal aortic compressor finds its greatest utility (Fig. 6).[8] The compressor is placed on the aorta against the lumbar spine in the lesser sac, superior to the stomach

Figure 6. The aortic compressor (Pilling Corporation) finds greatest use in control of hemorrhage from ruptured aortic aneurysms but can be employed in a number of ways in other forms of exsanguination.

and inferior to the liver. This allows a temporary period of suprarenal aortic occlusion, which permits maximal perfusion to the brain and coronary arteries while the surgical team isolates the infrarenal aorta above the aneurysm and cross-clamps in this location.

In approximately 15 per cent of patients, there will be free intraperitoneal hemorrhage, either from primary rupture of the aneurysm into the peritoneal cavity, or secondary rupture of the retroperitoneal hematoma into the peritoneum. Regardless of the extent of the hematoma, control of the aorta can be attained rapidly because of the preliminary dissection achieved by the hematoma. It is our philosophy to offer surgical intervention to any patient who can be brought to the operating room alive, since no patient will survive who is not operated upon.

Once the aortic clamp is in place, the operation can proceed as in the elective procedure. That is, the transverse colon is retracted superiorly and the small bowel superiorly and to the right. The patient's clinical situation is stabilized as plasma and blood are infused rapidly. It is at this time that 80 mg of furosemide is given with 12.5 gm of mannitol in order to minimize renal damage produced by shock.

It must be remembered that in most patients the diagnosis of a ruptured abdominal aneurysm can be made quickly by interpretation of the patient's symptoms and physical findings. The survival of the patient depends upon proper application of a vascular clamp to the aorta above the aneurysmal rupture. This can only be accomplished when the patient is taken to the operating room and not to the intensive care unit. An extensive work-up with consideration of the patient's other medical problems must await the survival of the patient after proper resection of his ruptured aneurysm.

POSTOPERATIVE COMPLICATIONS AFTER EMERGENCY RESECTION

It is of paramount importance to anticipate that postoperative respiratory insufficiency may develop if the patient survives emergency aortic resection. Pre-existing lung disease will contribute, as will preoperative smoking, prolonged anesthesia, and transfusion of large amounts of whole blood. The latter is due to passage of minute emboli through the usual commercial transfusion filters. Further, intravenous overloading is common during emergency aneurysm resection. This can produce a pulmonary edema which in itself is not significant but which, when added to the various other causes of pulmonary insufficiency, may represent a fatal additional impairment.

It is wise to leave the endotracheal tube in place until the patient is fully awake and postoperative blood gas values can be obtained. The tube may usually be removed within the first few hours after emergency aneurysm resection. After 48 hours, if an endotracheal tube is still necessary, one must consider placement of a tracheostomy, despite the attendant possibilities of endotracheal infection.

In the postoperative period, extensive use is made of positive pres-

sure ventilation, using humidified air and ultrasonic nebulization. Diuretics may be employed to mobilize excessive fluid during this period of time. If reduced pulmonary compliance is present, a much higher pressure will be required to deliver the necessary minute volumes of ventilation.

Fortunately, problems of electrolyte imbalance are uncommon in patients with aortic surgery; however, renal failure is to be anticipated in many patients who have suffered preoperative hypotension. In patients with aortic operations, the hourly urine output is a cornerstone of management. A desirable range of 30 to 50 ml per hour is maintained at all times. Oliguria may be due to acute tubular necrosis, severe hypoxia or development of metabolic acidosis.

As oliguria appears, the surgeon must assess the total volume load being presented to the kidneys and evaluate the need for diuretics. Two hundred ml of fluid is infused rapidly while the central venous pressure is watched. If a rise in central venous pressure occurs, the surgeon is assured that an adequate blood volume is present. Furosemide, 40 to 80 mg, is given immediately, and this is supplemented with sequential administration of furosemide and mannitol in order to keep the urine volume at a rate greater than 50 ml per hour. If urine output tapers off hour by hour, acute tubular necrosis is likely present. However, when urine output ceases abruptly, having previously been adequate, one must consider that the catheter itself has become obstructed. If optimal blood and fluid replacement have been achieved and diuretics are producing no significant change in the oliguria, one must assume that acute tubular necrosis has developed and fluid intake from this point on must be restricted to 600 ml. per day. The patient must be weighed daily and examined repeatedly for signs of circulatory congestion. Careful monitoring of serum electrolytes will give adequate warning that serum potassium is rising. If this does rise greater than 5.0 mEq/L, Kayexalate enemas must be given repeatedly in order to keep the potassium level within bounds. Hemodialysis may need to be instituted if the blood urea nitrogen level approaches 150 mg per 100 ml or the serum potassium level approaches 6.5 mEq/L. Peritoneal dialysis should never be performed in patients after aortic surgery.

SPECIAL PROBLEMS IN AORTIC ANEURYSM SURGERY

Since the abdominal aortic aneurysm is relatively common, a number of associated conditions arise in coincidence with this lesion. These present fascinating clinical considerations for surgical judgment.

Cholelithiasis is one of the most common associated conditions found at elective abdominal aortic aneurysm resection. While it is possible to take the viewpoint that only the aortic aneurysm should be treated at this time, it is a fact that an uncomplicated cholecystectomy can be performed in a very few minutes with a high degree of safety with the exposure given to the surgeon who is resecting the abdominal aorta. Therefore, our own considerations are as follows: If the aortic aneurysm resec-

tion has been smooth and there has been a minimum amount of retroperitoneal bleeding, if the retroperitoneum can be closed securely and tightly prior to turning attention to the gallbladder, if there are few adhesions to the gallbladder and the region of the common bile duct, and if the gallstones themselves are such that there are no indications for common duct exploration; then, the surgeon may elect to do a cholecystectomy.

When this is done, the conventional subserosal cholecystectomy is done without common duct exploration. A Penrose drain is placed in the hepatorenal fossa led through a subcostal stab wound. The cholecystectomy is done, of course, only after the retroperitoneum is closed and the viscera replaced over the region of the aneurysm. In no case should an elective appendectomy be performed.

Coincidental malignancy may be found during the abdominal exploration phase of the operation prior to resecting an abdominal aortic aneurysm. When this occurs, the malignancy takes precedence over the aneurysm in consideration for resection. This is because tumor seeding may occur and indeed may be occurring at the time of the operation, and it is necessary to minimize the chance for tumor spread if the patient is to achieve surgical cure. The patient will have an inevitable period of immunologic hyporesponsiveness in association with the surgical event.

Carcinomas of the stomach and colon, if resectable, should be removed at this time. This is true for relatively small carcinomas of the antrum of the stomach, and for right colon adenocarcinomas as well as those of the transverse and descending colon. Only if an unsuspected carcinoma of the rectum is found should an operation for cure of this lesion be deferred until the aneurysm itself is resected. This is because rectal carcinomas will necessitate abdominoperineal resections with employment of an end colostomy. It is far better to have a colostomy done after the aortic graft is in place for a period of time. Therefore, if rectal carcinoma is encountered, the aneurysm is resected, the retroperitoneum closed, and the abdominal wall closed. After a week, the patient is prepared and an elective abdominoperineal resection performed. Preoperative radiation can be given if indicated in the intraoperative interval.

While precedence is given to the carcinoma in the usual situation of elective aneurysm resection, if the aneurysm is quite large, it requires treatment prior to turning attention to the malignancy. That is, if the aneurysm is larger than 8 cm in diameter, it acquires precedence in treatment because the chance for its rupture in the postoperative period is too great.

In a reported series of 803 cases of aortic resection, Szilagyi[34] found 31 cases of associated malignant tumors. In one of these, a gastric carcinoma was found and a gastric resection performed. The large abdominal aortic aneurysm was not resected. It ruptured 3 hours postoperatively, and the patient did not survive attempted resection.

Thoracoabdominal Aneurysms

Abdominal aortic aneurysms extending to involve the renal arteries, superior mesenteric arteries and celiac axis are not for the casual opera-

tor. These are among the most difficult problems encountered by vascular surgeons, and are serious and challenging not just because of the extensive nature of their vascular involvement but also because of their anatomic inaccessibility. The first major consideration in therapy of these lesions is adequate exposure of normal aorta proximal and distal to the lesion. Only with proper exposure can attention be turned to the second major consideration: proper vascularization of viscera during aneurysm resection and the aortic reconstitution. Without going further into detail, it will suffice to say that a thoracoabdominal incision is required, and a Dacron tube graft is utilized as not only a temporary shunt around the aneurysm but also as the permanent prosthesis to which branch grafts can be attached for the various visceral arteries.

Aortocaval Fistula

Approximately 1 per cent of abdominal aortic aneurysms will rupture into the vena cava. Such an occurrence is characterized by high output cardiac failure, and physical findings so striking that diagnosis of this condition is simple. The abdominal aortic aneurysm is usually palpable and a thrill and murmur on the right side of the abdomen are present. A very low diastolic blood pressure confirms the diagnosis of arteriovenous fistula. Furthermore, coldness, swelling and cyanosis of the lower extremities is present due to decreased arterial perfusion and increased venous pressure. If the shunt is large, 1.5 cm or more in diameter, there will be a rapid deterioration in cardiac performance, with subsequent rapid renal and hepatic failure.

In approaching such a fistula surgically, one should take great care that aneurysmal contents of laminated clot are not discharged directly into the vena cava, causing fatal pulmonary embolization. It is sufficient merely to isolate the aorta above the aneurysm and the iliac vessels below, clamping these, opening the aneurysm widely and rapidly, and occluding the fistulous opening into the vena cava with a finger. Suturing of the caval wall is performed from within the aneurysmal sac. Clamping of the vena cava in such a situation is ill-advised, since right heart filling could be markedly impaired.

Aortoduodenal Fistula

While erosions of aortic aneurysms into the gastrointestinal tract are rare, they do occur. It is important to recognize that the gastrointestinal bleeding which results in such a situation may be intermittent and occult. An initial hemorrhage may terminate spontaneously, only to recur in hours or days. Massive hemorrhage will ultimately eventuate. Operation in such situations should be performed expeditiously and appropriate care should be given to sterilizing the operative field in the region of the aortoduodenal fistula after closure of the duodenum.

Horseshoe Kidneys and Aortic Aneurysms

There is no reason to expect that coincident horseshoe kidney and abdominal aortic aneurysm is more than a chance phenomenon. The fact is that they are found together, and this means that surgery for resection of the aortic aneurysm is interesting if not difficult. While there have been

fewer than 30 cases reported of abdominal aortic aneurysms associated with horseshoe kidney and only three of these have been in patients in which the aneurysm has ruptured, no doubt there are many unreported cases.

Horseshoe kidneys in general, and those associated with aortic aneurysms in particular, have lower pole fusion in 90 to 95 per cent of the cases. The calyces are usually rotated medially, the pelves anterior to the renal pedicles. The ureters are usually two in number, and these pass anterior to the isthmus. There may be from 1 to 10 arteries supplying the renal mass. In general, these fall into three major groups. The first is one major vessel to each pole of each kidney arising from above the aneurysm. The second is only one vessel supplying the kidney arising above the aneurysm, the remainder arising from below the aneurysm. The third category is no major artery arising above the aneurysm and all vessels taking off from the aneurysm below. Each circumstance requires its own method of revascularization.

Ordinarily, venous drainage is no problem unless common venous drainage emptying into the vena cava at a higher than normal level is mistaken for an inferior mesenteric vein. In principle, extreme care must be used to protect the entire vascular supply and the collecting system of the renal complex. If possible, an arteriogram should be performed if a fused kidney is suspected. However, if a horseshoe kidney is encountered unexpectedly during operation for an abdominal aortic aneurysm, it may be worthwhile to perform an operative arteriogram if the aneurysm itself has not ruptured. Time should then be taken to dissect out all anomalous vessels and to reanastomose all of the arteries if possible. It may be that portions of kidney will be ischemic following aortic resection. If so, resection of the ischemic portions of kidney must be undertaken before the abdomen is closed.

Aortic Aneurysm Resection in the Aged

There have been a number of reports of aneurysm resection in patients over 80 years of age. Edmunds has provided the most thoughtful assessment of this problem.[14] He concludes that operation is advisable in the octogenarian if rupture of the aneurysm has occurred or if there is a recent onset of symptoms, and if patients are free of serious disease. In the ruptured aneurysm situation, immediate operation is necessary unless the patient is moribund from other disease. Whether or not operation is advisable for large aneurysms alone or for chronically symptomatic aneurysms is debatable, and operation probably should not be attempted if the patient has serious associated chronic disease. Certainly, operation for small asymptomatic aneurysms in octogenarians should not be advised in view of the relatively short average life expectancy of such patients.

SUMMARY

Modern management of aortic aneurysms is characterized by a direct approach to the problem. Factors which influence surgical judgment

include knowledge that the condition is common, lethal and unpredictable. It is unfortunate that these lesions arise in an aged population which harbors many associated conditions. The most common of these may also be dealt with expeditiously.

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Aorto-Iliac Occlusive Disease

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The surgical management of aorto-iliac occlusive arterial disease is well established, although there remains a variation in operative procedures advocated by different surgeons.[4, 5, 6, 9, 12, 15] There is general agreement that localized aorto-iliac disease can be effectively treated by endarterectomy and extensive aorto-iliofemoral disease is usually best treated by bypass grafting procedures, particularly in the elderly poor risk patient. Many patients fall between these two extremes and the operative procedure must be fitted to the individual patient. The surgeon's personal experiences and biases will naturally influence his choice of operative reconstruction. This communication is an effort to emphasize some of the diagnostic and therapeutic problems in the management of patients with aorto-iliac occlusive disease.

ANGIOGRAPHIC ASSESSMENT

The need for excellent angiography in all investigative procedures is well recognized. Not infrequently, the extent of occlusive disease is underestimated by angiography because of the segmental and eccentric nature of atheromatous lesions.

Case 1. A 47-year-old white woman presented with a 6 month history of intermittent claudication in both legs. The femoral pulses were slightly reduced, but the pedal pulses were all present although reduced in volume. There was a midabdominal bruit which radiated to both lower quadrants. The right ankle systolic pressure was 100 mm Hg with a brachial pressure of 137/76, and the left ankle systolic pressure was 93 mm Hg with a brachial pressure of 138/81. This arm to ankle pressure gradient at rest indicated a significant main artery obstruction to both lower limbs. Translumbar aortography demonstrated a very localized defect in the midabdominal aorta (Fig. 1*A*) with a normal distal tree. The severity of the stenosis was much more apparent on the lateral aortogram (Fig. 1*B*), which demonstrated a posterior plaque with a diaphragm type of narrowing. Aortic endarterectomy was performed with restoration of normal circulation and relief of symptoms.

Case 2. A 48-year-old white man had a 1 year history of intermittent claudication in both legs affecting the calf, thigh and buttock. The left femoral pulse was

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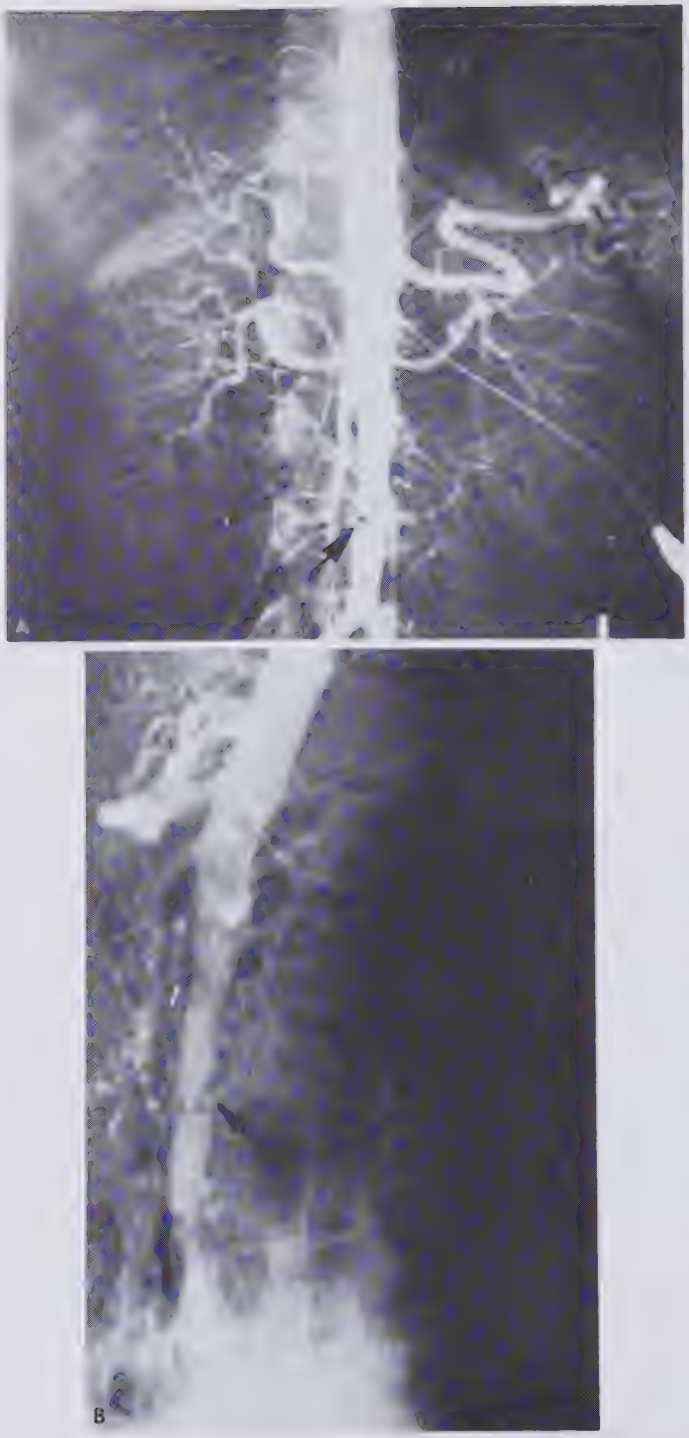


Figure 1. *A*, Case 1, translumbar aortogram: arrow denotes transverse narrowing in midaorta. *B*, Lateral projection: arrow shows severe stenosis on posterior wall of midaorta.

graded 1/4 and the left dorsal pedis 2/4. The right leg pulses were all graded 4/4. On auscultation there was a bruit in the right iliac fossa and also over both common femoral arteries. The right ankle systolic pressure was 135 mm Hg with a brachial pressure of 125 mm Hg. The left ankle pressure was 90 mm Hg with a brachial pressure of 125 mm Hg, indicating significant obstruction in the arteries supplying the left leg. A translumbar aortogram suggested stenosis in both iliac arteries on the anteroposterior exposure (Fig. 2*A*). These lesions were much better demonstrated on the oblique exposure, which showed almost complete obstruction in the left common iliac artery and mild stenosis on the right (Fig. 2*B*). An aorto-bilateral common iliac endarterectomy extending to the middle of both external iliac arteries was performed. The postoperative ankle pressure was 140 mm Hg on the right and 130 mm Hg on the left, with a brachial pressure of 130 mm Hg.

These two patients demonstrate that single-plane angiography may underestimate the extent of occlusive disease present. The arterial pressure measurements are sometimes helpful in suggesting the presence of an occlusive lesion, although the resting ankle pressure may be normal when symptomatic disease is present, as shown in Case 2.

ACUTE ISCHEMIA WITH AORTO-ILIAC OCCLUSIVE DISEASE

About 10 per cent of patients with symptomatic aorto-iliac occlusive disease present with symptoms and signs of acute ischemia. Distal embolic occlusion may be associated with proximal stenotic or occlusive lesions of the aorta or iliac arteries[3, 13] causing severe ischemia.

Case 3. A 64-year-old white man was admitted to hospital 10 days after a minor injury to his right foot. There was no history of intermittent claudication. The right great toe was cyanosed and cool with diminished sensation. The forefoot was erythematous but cool. The femoral pulses were recorded as 4/4. The right popliteal pulses were normal, but no pedal pulses were present on the right. All distal pulses were normal in the left leg. A right femoral arteriogram demonstrated a thrombus in the right common iliac artery and occlusion of the anterior tibial and posterior tibial arteries near the ankle (Fig. 3). A translumbar aortogram (Fig. 4) showed bilateral iliac stenosis with a filling defect in the right iliac artery which was probably thrombus. An aorto-bilateral iliac endarterectomy was performed.

At operation the thrombus was superimposed on an ulcerated stenotic atheromatous plaque (Fig. 5). The distal end of the thrombus was fractured, suggesting that a segment had broken off and embolized distally. A right lumbar sympathectomy was also performed. Gangrene limited to the right great toe developed (Fig. 6), and a toe amputation healed successfully. The occlusions remained in the tibial arteries, but satisfactory collateral circulation developed to preserve function in the remainder of the foot.

Case 4. A 65-year-old white woman was admitted with a diagnosis of acute arterial occlusion in the left leg in May 1965. She had had intermittent claudication in the left leg affecting the calf, thigh and buttock for 6 weeks. Twenty-four hours before admission the left leg became cold, cyanosed and numb. The left anterior compartment muscles were tender and the foot was anesthetic. No pulses were palpable in the left leg, but all pulses were normal in the right leg. An aortogram was done with a catheter through the right femoral artery. There was an occlusion of the left common iliac artery, with excellent collaterals filling the distal arteries and an embolus in the left popliteal artery (Fig. 7*A* and *B*). A left common iliac thromboendarterectomy and left lumbar sympathectomy were performed. A popliteal embolus was removed directly with a medial approach to the popliteal artery. The postoperative angiogram demonstrated a patent distal popliteal artery and branches (Fig. 7*C*). A fragment of the soft thrombus associated

(*Continued on page 203*)

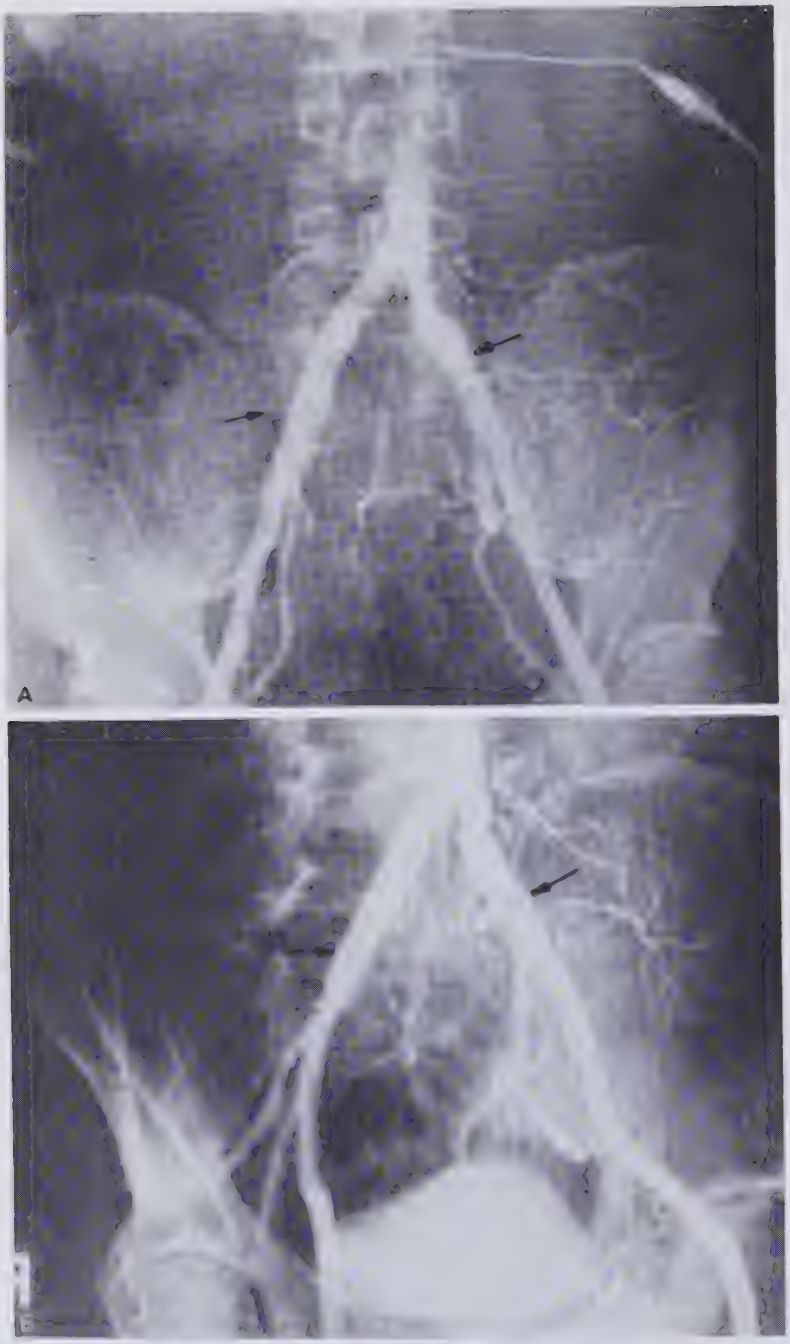


Figure 2. *A*, Case 2, translumbar aortogram, anteroposterior projection: arrow denotes suspected sites of stenosis in common iliac arteries. *B*, Translumbar aortogram, oblique projection: arrow denotes sites of stenoses in both common iliac arteries.

Figure 3. *A*, Case 3, femoral arteriogram showing filling defect in the right common iliac artery. *B*, Angiogram showing occlusion of anterior and posterior tibial arteries.

Figure 4. Case 3, translumbar aortogram showing bilateral common iliac artery stenosis and thrombus in right common iliac artery.

Figure 5. *A*, Case 3, operative photograph showing thrombus in common iliac artery with fractured distal end. *B*, Operative photograph after removal of thrombus. Note ulcer in artery at site of stenosis.

Figure 6. Case 3, gangrene in right great toe.

Figure 7. Case 4, aortogram showing left common iliac occlusion. *B*, Embolus in left popliteal artery. *C*, Post repair angiogram showing patent popliteal artery.

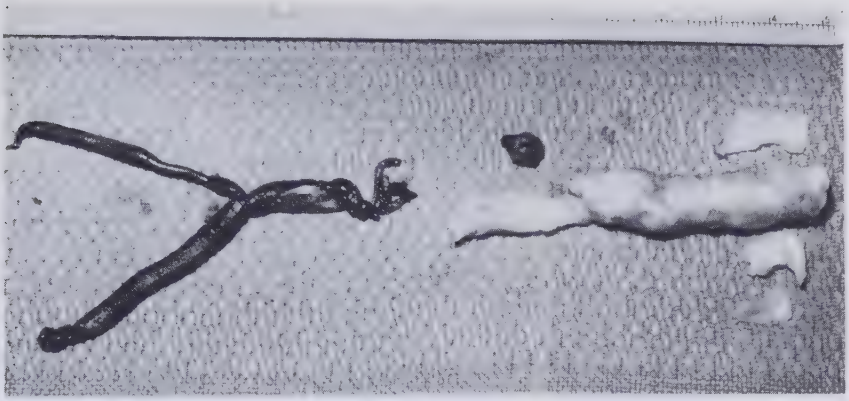


Figure 8. Case 4, thromboendarterectomy specimen and thrombus. The small fragment was removed from the popliteal artery.

with the atheromatous plaque probably embolized to the popliteal artery (Fig. 8). The postoperative course was uneventful and she remains asymptomatic 8 years later. The postoperative ankle pressures were 125 mm Hg with a brachial pressure of 135 mm Hg.

These two patients demonstrate the occurrence of distal embolic occlusion associated with aorto-iliac occlusive disease. The first patient (Case 3) illustrates the importance of proximal angiography even when the femoral pulses are clinically normal. Recurrent distal occlusions can be prevented by adequate treatment of the proximal iliac lesion. The second patient (Case 4) demonstrates the importance of complete visualization of the distal tree even when the proximal lesion is obvious. Failure to remove the popliteal embolus may have resulted in limb loss or at least persistent ischemia.

ALTERNATIVES TO AORTO-ILIAC RECONSTRUCTION

Axillofemoral Bypass

Since the introduction of the axillofemoral bypass by Louw[7] and Blaisdell[1] for aorto-iliac disease, it has met with variable degrees of success. Mannick[8] recently reviewed his experience and expressed the opinion that axillo-femoral bypass may have long-term function in some patients but should be reserved for poor-risk patients. We have recently reviewed our own experience with 16 axillofemoral grafts in 14 patients. One patient had bilateral grafts for infection of an aortic prosthesis. This patient died postoperatively of a stroke with patent grafts. The remaining 14 grafts have been done for end-stage ischemia with rest pain or gangrene in poor-risk patients. There have been five early occlusions and five late occlusions. Four of the late occlusions occurred within a year of operation. One graft remained patent for 30 months and then occluded.

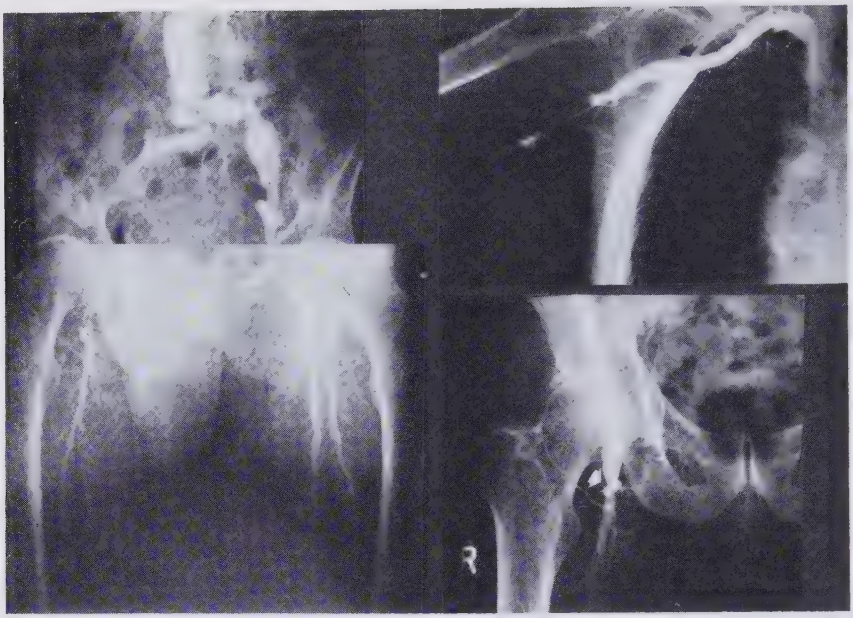


Figure 9. Case 5, (*left*) translumbar aortogram showing a small aneurysm, right iliac occlusion, and bilateral superficial femoral occlusions. (*Right*) Axillofemoral Dacron graft.

Two patients presently have patent grafts and both are less than a year since operation. We have not used the axillo-bilateral femoral graft, as has been suggested by Moore[10] to improve the long term patency rate.

Case 5. A 70-year-old white man had rest pain in his right foot for 3 years. He had a history of myocardial infarction and pulmonary disease. Translumbar aortography demonstrated a right iliac occlusion, bilateral superficial femoral occlusions, and a small aortic aneurysm (Fig. 9). Because of his general cardiopulmonary status and the angiographic findings, he was considered an ideal candidate for an axillofemoral bypass graft. A 10 mm knitted Dacron graft was inserted (Fig. 9). The rest pain was relieved, but he died 6 months later of a myocardial infarction.

This patient was considered an ideal candidate for an axillofemoral graft because of his cardio-pulmonary disease and the magnitude of the direct operative procedure dictated by the presence of the aneurysm. We think the procedure should be reserved for patients with aortic prosthetic sepsis or for patients who are poor operative risks for a conventional approach and have end-stage ischemia, but warrant an attempt at limb salvage.

Cross Femoral Bypass

The cross femoral graft was introduced by Vetto[17] in 1962. It is used primarily in poor-risk patients with unilateral iliac disease.

Case 6. A 60-year-old white man presented with rest pain in his left foot. He had experienced intermittent claudication in the calf and thigh for 9 months and

rest pain for 1 week. The systolic pressure at his left ankle was 50 mm Hg with a brachial pressure of 133/86 mm Hg. The right ankle pressure was 113 mm Hg with a brachial pressure of 125/91. This suggested that there was also disease in the right leg, although it was asymptomatic. The right femoral pulse was considered weak on palpation, but the patient was markedly obese. A translumbar aortogram demonstrated an occluded left iliac system, but there was no demonstrable disease in the right iliac arteries (Fig. 10*A*). Because of his obesity and angina pectoris, a cross femoral graft was advised. At operation the donor femoral artery pressure was measured by strain gauge and found to be 125/70 mm Hg with a brachial pressure of 122/78 mm Hg. The left common femoral artery pressure was 60 mm Hg. In view of the normal right femoral artery pressure a cross femoral graft was performed. His postoperative course was uneventful except for urinary retention. The postoperative ankle pressures were equal and only slightly below normal.

Although cross femoral grafting has been reserved for poor risk patients by most surgeons, Mannick[8] and Brief et al.[2] have suggested that the indication be extended to include good risk patients with unilateral disease. The reluctance to perform cross femoral grafts as a preferred procedure is based on the presumption that iliac disease on the donor side will progress and cause occlusion. Minken et al.[9] have reported that the results with unilateral repair are comparable to the results of bilateral repair in aorto-iliac disease. In our own series of 149 unilateral reconstructions for iliofemoral occlusions, 12 patients or 8 per cent have come to bilateral repair. The average time from unilateral repair to bilateral reconstruction was 36 months. These findings suggest that cross femoral grafts may be a satisfactory alternative for unilateral iliac obstruction. It has been demonstrated that proximal disease on the donor femoral side will adversely affect flow to both legs.[16] In addition to adequate preoperative angiography and bilateral leg arterial pressure measurements, intraoperative femoral artery pressure measurements are strongly advised before proceeding with cross femoral grafts.

COMPLICATIONS OF AORTO-FEMORAL GRAFTS

Although early and late complications of aorto-iliofemoral reconstruction are not common, when they occur, they may be catastrophic.

Groin Infection

The gravest complication confronting patients subjected to arterial reconstructive procedures is infection in the operative site. The commonest site of infection is in the groin incision in patients subjected to aortofemoral bypass prosthetic grafts.

Case 7. A 58-year-old white man was admitted in October 1967 with ischemic rest pain in his left foot. He had suffered from bilateral calf claudication for 4 months and rest pain for 1 month. Translumbar aortography demonstrated bilateral superficial femoral artery occlusion and left iliac occlusion. The systolic pressure at the left ankle was 60 mm Hg and at the right ankle 80 mm Hg, with a brachial pressure of 225/110 mm Hg. A left common iliac to common femoral Dacron bypass graft was inserted as well as a femoropopliteal reversed autogenous vein graft. Postoperative infection developed in the left groin which required drainage and debridement. He was discharged with a patent graft (Fig. 11*A*) and had intermittent drainage from the wound. Six months later he presented with re-

Figure 10. *A*, Case 6, translumbar aortogram showing left iliac occlusion. *B*, Delayed filling of common femoral and branches.

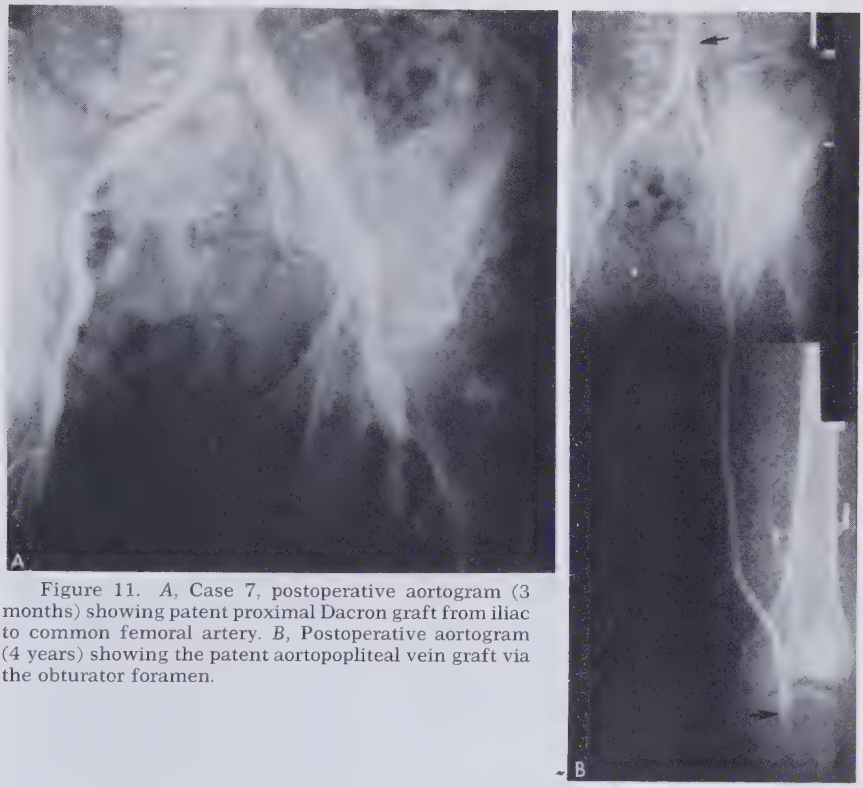


Figure 11. *A*, Case 7, postoperative aortogram (3 months) showing patent proximal Dacron graft from iliac to common femoral artery. *B*, Postoperative aortogram (4 years) showing the patent aortopopliteal vein graft via the obturator foramen.

current infection and a false aneurysm at the anastomosis. Another vein graft was inserted from the aorta, through the obturator foramen to the previously placed vein graft. The groin wound was excised and the prosthesis removed. The graft continued to function 4 years later, although the patient had minor claudication in both legs (Fig. 11*B*). The left ankle pressure at this time was 223 mm Hg, with a right brachial pressure of 224/115. The right ankle pressure was 75 mm Hg.

Case 8. A 53-year-old man had claudication in both legs for 1 year and rest pain in the right foot for 3 weeks before admission to hospital. Aortography demonstrated right common iliac stenosis and right external iliac occlusion (Fig. 12*A*). There was occlusion of both superficial femoral arteries. A right common iliac to common femoral prosthesis was inserted after endarterectomies at both anastomotic sites. Purulent drainage occurred from the groin wound postoperatively, but it appeared to heal. The postoperative ankle pressures were 85 mm Hg in both legs with a brachial pressure of 128/82 mm Hg. Eleven months later the patient returned with infection and a sinus in the right groin, although the wound had appeared normal 6 months previously at his routine postoperative follow-up visit. A sinogram was done and outlined the prosthesis to the proximal anastomosis (Fig. 12*C*). The prosthesis was removed, with repair of the common iliac artery and closure of the common femoral artery. Fortunately there was excellent collateral circulation and his limb survived. He has claudication at 75 yards but is able to do his work.

Groin infections can be disastrous and must be avoided. In most instances it is probable that contamination occurs at the time of surgery, even when the infection does not present until late postoperatively. It is

Figure 12. *A*, Case 8, preoperative aortogram showing the occlusion of the right iliac and both superficial femoral arteries. *B*, Postoperative aortogram showing functioning graft from common iliac to common femoral artery. *C*, Sinogram showing contrast medium extending from groin to proximal anastomosis and surrounding the prosthesis.

usually necessary to remove the prosthesis completely before the infection is eradicated. Although the infection appeared localized in Case 8, the sinogram demonstrated its true extent along the full length of the prosthesis. Meticulous hemostasis and aseptic technique are mandatory in vascular surgery.

Anastomotic Aneurysm

Anastomotic false aneurysms occur because of disruption of the anastomotic suture line or weakening of the adjacent arterial wall.[14] Associated low-grade infection may contribute to this complication. Aneurysms usually occur when silk sutures have been used; however, they may also occur when Dacron suture is used. Local endarterectomy is frequently necessary in the common femoral artery to ensure a good outflow, and this may predispose to arterial wall disruption.

Case 9. A 60-year-old white man had intermittent claudication in both legs for 5 years. He presented with rest pain in the left foot for 1 month. The ankle pressures were 45 mm Hg when the brachial pressure was 108/80. Aortography demonstrated a small abdominal aortic aneurysm with iliac aneurysms and bilateral superficial femoral artery occlusions. An aorto-bilateral common femoral Dacron prosthesis was inserted. The aneurysm was excluded and endarterectomies were done in both common femoral arteries prior to the anastomoses, which were performed with 5–0 braided Mersilene. The postoperative angiogram was satisfactory. The patient returned 3 years later with occlusion of the left limb of the graft. There was a nonpulsatile mass in the left groin and a pulsatile mass in the right groin. The aortogram demonstrated the right femoral anastomotic aneurysm and occlusion of the left limb of the prosthesis (Fig. 13). The right ankle pressure was

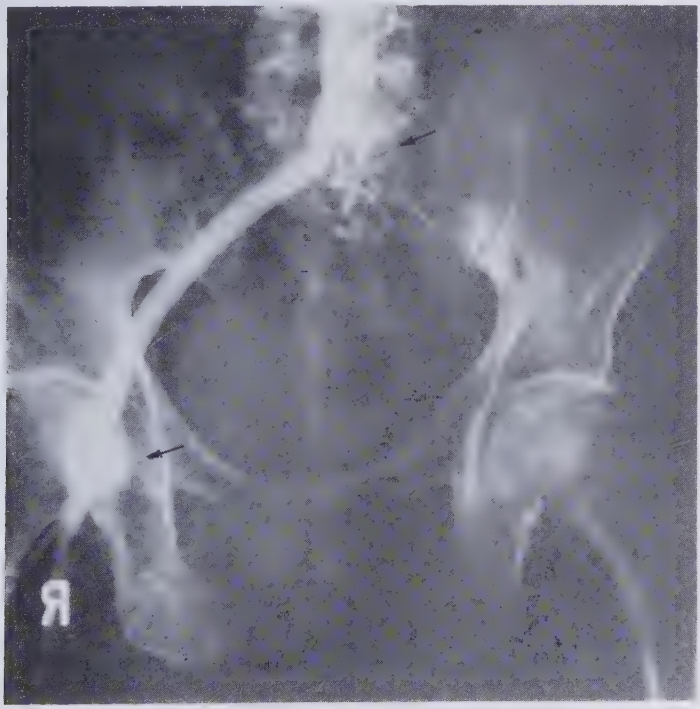
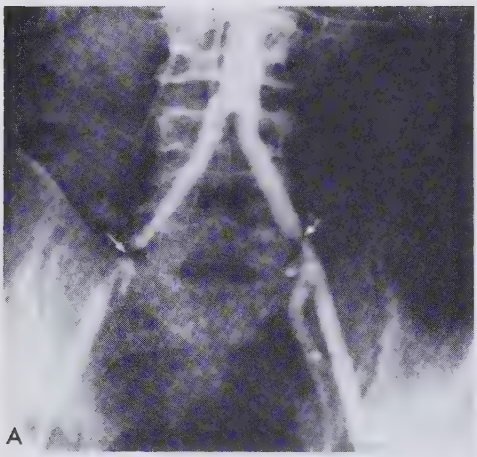


Figure 13. Case 9, anastomotic right femoral aneurysm and occluded left limb of the graft.



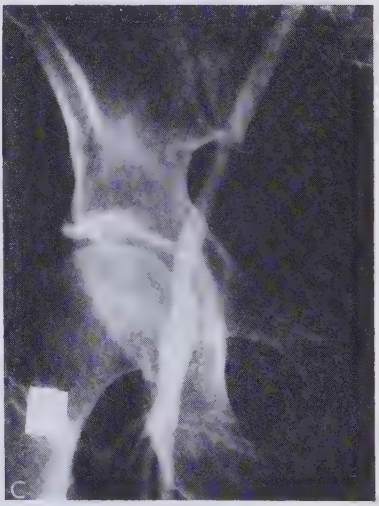


Figure 14. *A*, Case 10, prosthetic Dacron graft in place 12 months with bilateral anastomotic stenosis. *B*, Anastomotic stenosis in right common femoral artery at 13 months postoperative. *C*, Postoperative vein patch angioplasty of anastomotic stenosis.

95 mm Hg and the left ankle pressure was 42 mm Hg when the brachial pressure was 114/84. Both false aneurysms were repaired with extensions added to the previous prosthetic limbs. The distal anastomoses were then done to the profunda femoris arteries after division of the origin of the superficial femoral arteries. The grafts have functioned satisfactorily for 2 years.

The cause of the false aneurysms was not apparent in this patient, since Mersilene sutures had been used and there was no evidence of infection. Anastomotic aneurysms are not common and fortunately, when not associated with infection, are usually readily repaired. Although endarterectomy may weaken the wall of the host artery and predispose to anastomotic aneurysm, fear of this complication should not preclude endarterectomy to provide a good outflow.

Anastomotic Stenosis

Late occlusion of aortofemoral prosthetic grafts is usually due to progression of distal disease with outflow occlusion. Anastomotic stenosis due to local thrombus may lead to graft occlusion in the absence of progressive disease or a demonstrable technical error.

CASE 10. A 39-year-old white man was first seen in March 1966 with intermittent claudication in the right calf. Aortography demonstrated right common iliac artery stenosis. An endarterectomy was performed, which occluded within 1 year. An aorto-bilateral iliac Dacron prosthesis was then inserted, which developed anastomotic stenosis bilaterally within 1 year (Fig. 14*A*). Both limbs were extended to the external iliac arteries on two occasions. The last graft to the right common femoral artery stenosed about 1 year later (Fig. 14*B*), causing a recurrence of claudication. This stenosis was repaired using a vein patch at the anastomosis (Fig. 14*C*). Serial ankle pressure measurements have suggested the presence of stenosis before occlusion has occurred, and this has led to early repair before graft occlusion. The stenotic lesions have been unorganized thrombus at the anastomosis on each occasion. There has not been any progression of distal disease over the 7 years of observation, and there is no evidence of hyperlipidemia or diabetes mellitus. An undetermined hypercoagulable factor predisposing to this anastomotic thrombus is suspected but unproved.

SUMMARY

Some of the major problems in management of aorto-iliac occlusive disease are discussed. The need for complete and multiple plane angiography is demonstrated. Physiological assessment by pressure measurements as suggested by Mozersky et al.[11] allow better assessment of the progression of disease and recurrence of stenotic lesions. The most important postoperative complications are also presented and methods of management outlined by illustrative case histories.

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Future Directions in the Development of Arterial Prostheses for Small and Medium Caliber Arteries

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ARTERIAL PROSTHESES: PROBLEMS AND DIRECTIONS

The majority of deaths in the western world are due to impairment of arterial flow to distal tissues. Since the most practical method for increasing distal flow is the bypass graft, the clinical need for arterial prostheses is virtually unlimited. Though prostheses may never be developed that will be equal to suitable autologous vascular grafts, certainly ones will be found that will be superior to inadequate saphenous veins or inadequate internal thoracic or radial arteries.

Unfortunately, all currently available prostheses have technical limitations (stiffness, thickness and poor conformability) which render them unsuitable for small arteries. Furthermore, the older, more conventional types of arterial prostheses exhibit very poor healing characteristics in man.[1, 4, 18] While the newer types of prostheses[9] have improved healing

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characteristics, their physical properties are no better than those of the older types.

Obviously, the primary concern of artificial grafts is maintenance of a patent channel without embolic complications. Conventional prostheses, developed over the last 20 years, are frequently successful in large arteries, despite poor healing, over long periods of time. But, because of poor healing, these grafts—pervious tubes of knitted fabric such as Dacron—are constantly subject to the danger of thrombotic occlusion. The flow surface is highly thrombogenic, since at implantation the prosthesis reacts with blood to form a fabric-thrombus complex. The interstices are closed by thrombus and all surfaces of the fabric are covered by thrombus. Even over a period of many years, such grafts fail to make significant progress in healing. The generally poor suturability and conformability of conventional grafts increase the predisposition to occlusion.

With existing prostheses, we believe that the best assurance of long term patency without complication is attainment of complete healing of the prosthetic wall, which includes an endothelialized flow surface. This final healed state is principally dependent upon the ingrowth of areolar tissue from perigraft sources through the interstices of a pervious graft wall.

The reason for emphasis upon complete healing of the prosthesis is that blood clotting characteristics of the flow surface become relatively normal only with the development of an endothelialized tissue surface, and thus protection from sudden thrombotic closure occurs only with complete healing.[19]

We feel that rapid attainment of this healed state will be the key to success in small arteries such as the coronaries or those in the lower leg, where conventional prostheses cannot be used successfully because of thrombotic closure. Furthermore, a prosthesis with rapid healing characteristics could be used in medium caliber arteries (for instance, femoropopliteal bypass above or below the knee joint) with a greater degree of success than is possible with prostheses available today.

However, there is always the possibility that development of a superior impervious prosthesis could remove the need for healing of the inner surface. To be superior to an optimally healed pervious graft, however, a prosthesis impervious to cellular ingrowth must have a flow surface equally resistant to thrombus formation. Combined with improvements in handling properties and suture line stability, a superior flow surface could make the impervious prosthesis preferable, since its optimal characteristics would be present from the moment of implantation; by contrast, the optimal characteristics of the pervious graft are delayed until the healing process is complete. For the present, however, there is no impervious prosthesis as good as the conventional porous fabric prosthesis.

Porous fabric prostheses of woven or knitted construction have been the standard in the surgical field until the present. Woven grafts have low porosity and hence bleed very little at implantation, but they do not heal well precisely because of this poor porosity. Knitted grafts on the contrary

are far more porous and hence require pre-clotting at implantation, but they heal better. An inherent disadvantage of all knitted fabric prostheses is that they must react with blood to form an impervious fabric-thrombus complex, the surfaces of which are highly thrombogenic. Subsequent healing of the graft by ingrowth of tissue is really the organization of the thrombus portion of this fabric-thrombus complex.

Fabric prostheses can be used with a high degree of success in relatively high flow rate areas like the abdominal aorta and the aorto-iliac or aortofemoral regions, either as replacement or bypass grafts. But their use in the medium-sized arteries—such as for femoropopliteal bypass even above the knee joint—has been associated with a high incidence of delayed thrombotic occlusion. Hence, it is not surprising that grafts of this type are completely unsuitable for small arteries.

Man heals arterial prostheses very slowly in contrast to the dog, pig, calf, and particularly the baboon.[10] DeBakey et al.[4] found that the inner flow surface of porous arterial prostheses in man was seldom formed by tissue but rather by fibrin in most instances. Warren and McCombs[18] have reported similar observations.

Our own observations[1] of 22 prostheses implanted in man for an average of approximately 2 years, with one in place for 11 years, also showed flow surfaces lined by compacted fibrin (see Fig. 2*A*) except within a few millimeters of the anastomoses, where pannus ingrowth from the arterial attachment site formed the flow surface. Despite the limitations of incomplete healing, such prostheses are reasonably suitable conduits for large caliber arteries with high flow rates.

Nonetheless, we believe that long-term results with pervious grafts, even in such favorable implant sites, would be superior if complete healing could be attained. On the basis of our observations in dogs and baboons we suspect that endothelial cells can deposit from the bloodstream (fallout seeding) upon certain types of fibrin surfaces in man and proliferate to form the flow surface of the prosthesis. Investigation is required as to the attractiveness of different fibrin surfaces to endothelial colonization from fallout sources. DeBakey et al. documented the presence of patchy endothelium in man on the fibrinous lining of a 6 cm length of knitted Dacron prosthesis removed after 46 months.[3] We have seen endothelium covering the surface of a partially thrombosed femoral artery aneurysm in man.[1]

Accordingly, we suggest that porous prostheses with "smooth" walls function as well as they do because the whole blood thrombus lining their flow surfaces at implant converts with time to a fibrin lining of less thrombogenicity. Furthermore, an endothelial lining may develop upon this surface by fallout healing from bloodstream sources.[15]

In view of the foregoing problems, our discussion of the problems and developmental possibilities of prostheses is based upon the search either for a truly satisfactory artificial flow surface—a means to eliminate the healing process—or for means to accelerate healing by aiding the ingrowth of perigraft tissues and also, if possible, by using a healable, nonthrombogenic substance as the initial flow surface.

DESIGN CONCEPTS FOR IMPROVED ARTERIAL PROSTHESES

Eliminating the Healing Process: Impervious Prosthesis (Synthetic, Thrombus-Resistant Flow Surface)

A theoretical advantage of an artificial flow surface is that it would not need to be pre-clotted, and thus need not begin its work with an initial thrombus layer. Optimal characteristics would be present immediately, and problems delaying healing, such as perigraft hematoma, would not affect this kind of graft.

In order for an impervious prosthesis to be workable, however, it must meet five rigid specifications: (1) thrombus-resistant flow surface, (2) high degree of conformability, (3) ease of suturability, (4) outer surface to which surrounding tissue can adhere firmly, and (5) inner surface to which any thrombus that does form can attach securely to discourage embolization. The thrombus-resistant nature of the flow surface must be permanent, since the body is given no opportunity to create its own surface. Similarly, since perigraft tissue cannot grow through the graft, some means of adherence is required to prevent the graft from tearing loose and bleeding during unusual flexion or during thrombectomy should the graft occlude and need to be reopened.

Unfortunately, no material has yet been developed that satisfies all of these criteria. For example, we have evaluated Gore-Tex* (expanded polytetrafluorethylene) and found that it meets specifications 2 through 5, but not the all-important number 1.[14] Even in the thoracic aorta of the dog the flow surface of this material becomes covered by thrombus, though usually the layer is thin and quite uniform.

The surface of Gore-Tex is formed of innumerable tiny openings a few microns in size—too small to allow the perivascular tissues to grow deeply into the wall, but large enough to enable these tissues to penetrate superficially and cling to the outer surface. We presume that thrombus forms in the tiny openings of the flow surface. We suggest that these minute thrombi cause the flow surface to attract a thin, uniform thrombus deposit which is securely anchored to the wall by its "tentacles" extending into the openings.

We are hopeful that further research will lead to development of a material that satisfies all five criteria, thus providing a graft free of clotting problems from the time of implantation. A prosthesis constructed of such material would be invaluable for both large and small caliber vessels. However, unless a material is found that provides a truly thrombus-resistant flow surface, the best hope for better prostheses rests with fabric designs that will allow quicker and more complete healing than is possible with any prosthesis available today. A further advance, which would make such a pervious graft as good as an ideal impervious graft, would be development of a nonthrombogenic yet healable substance to replace thrombus in rendering the graft blood-tight.

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Accelerating the Healing Process: Pervious Prosthesis with Flow Surface Healed by Tissue Ingrowth

Virtually all prostheses in clinical use today are fabric with open interstices. These interstices must be closed by thrombus, either before or after implantation. Once they are sealed and the surfaces coated by whole-blood thrombus, blood and perivascular tissues make contact only with the thrombus surface. The prosthesis, porous before contact with blood, becomes a "blood-tight" fabric-thrombus complex permeable to cellular ingrowth.

The whole blood thrombus sealing the interstices of the prosthesis may thus be thought of as the ground substance of the prosthesis. Unfortunately, whole blood thrombus is exceedingly thrombogenic, and thrombus will continue to accumulate on the flow surface unless the velocity of blood flow is above a critical point which we term the "thrombotic threshold velocity" (see p. 223). A second disadvantage of thrombus as a ground substance is that the greater the amount of thrombus accumulated on the flow surface, the longer healing will be delayed. This is particularly true in man, who appears to have much less ability to organize thrombus in this location than does the pig, calf, dog or baboon.

Despite these negative features, thrombus does have certain practical advantages as the ground substance for a porous fabric prosthesis. It is universally available at operation by interaction of the graft with blood. Furthermore, thrombus binds tenaciously to a surface that has any degree of roughness at all, such as a porous fabric prosthesis—thus any thrombus that does form tends to remain attached unless it dangles in the moving bloodstream and fragments.

An important advantage of thrombus is its permeability to cellular ingrowth, allowing for the possibility of complete healing. Certainly, a nonthrombogenic ground substance is needed but it must at least equal thrombus in the security of its attachment and in its permeability to cellular ingrowth.

In contrast to whole-blood thrombus, fibrin, having a negative charge, is relatively nonthrombogenic.[12] Yates et al.[19] have measured the surface thrombogenicity of arterial prostheses after varying implantation times. They placed whole blood on the grafts and timed the first appearance of fibrin (surface clotting time, or SCT). At implant this was about 15 seconds and after several days to a few weeks was prolonged to 1 to 2 minutes. Little further prolongation of the SCT was then observed until complete healing of the inner wall had occurred, at which point the SCT approximated that of the normal aorta (15 minutes).

The early prolongation of the SCT appeared to be due to conversion of the whole-blood thrombus forming the flow surface to a fibrin type residual of lesser thrombogenicity; the later prolongation appeared to be due to the complete healing of the inner wall with the establishment of an intact endothelialized flow surface. (Though we now recognize the gross changes that take place in the graft surface, we know little of the reactions that produce these changes.)

Few clinical grafts attain a completely healed flow surface. Most are successful despite this—in large arteries. Probably their success is a

result of the development of a "good" fibrin lining that is moderately resistant to whole-blood thrombus deposition in these high-flow areas. However, the danger of thrombotic occlusion would be considerably less if complete healing occurred.

TRELLIS CONCEPT OF GRAFT WALL CONSTRUCTION. Perigraft tissues react to a prosthesis by forming a fibrous capsule around it. From this outer capsule, ingrowth extends through the interstices of the prosthetic wall to invade the thrombus layer forming the flow surface. If complete and excellent healing occurs, all thrombus is replaced by healthy fibrous tissue and the flow surface is formed by an intact layer of endothelium. The thrombogenicity of the flow surface is similar to that of a normal artery.[19]

There may be more than one way to encourage the tissue ingrowth. Polyurethane microporous materials are under investigation,[2] as are fabric constructions termed "nonwoven." We have found a helpful physical arrangement of the graft wall which provides a trellis for fibroblasts to "crawl" upon. We have termed this design the "trellis concept" of graft construction.

The "trellis" consists of a roughened fabric construction that differs significantly from the relatively "smooth" surface of the pervious prostheses in common use today. In the "smooth" grafts, the yarn is fashioned to form relatively uniform surfaces that face the blood on the inside and the perigraft tissues on the outside. The pores are rather crisply defined. This basic fabrication is in wide general use and is the one we refer to as "conventional" in design.

"Rough" graft construction is still evolving. These grafts are filamentous on one or both surfaces, and the interstices are more or less criss-crossed by synthetic fibrils. The inside or outside (or both) is "fuzzy" due to the presence of multiple separated yarn filament loops or ends. We began experimenting with rough grafts in the hope that, since fibroblasts migrate upon filaments, a filamentous surface would assist fibroblastic attachment and penetration of the wall more effectively than would a relatively smooth surface.

In our graft, we have employed velour to provide this filamentous configuration to the outer surface. The interstices are greatly subdivided by the monofilament velour strands. These filaments provide a finely divided three dimensional framework along which the fibroblasts can grow extensionally, as seen in tissue culture. The cross sectional views of a smooth-walled knitted prosthesis and of three different types of velour prostheses are shown in Figure 1.

Our clinical experience with 218 external velour knitted Dacron prostheses during the past 3 years (April 1970 to April 1973) includes 42 aortic bifurcation replacements, 81 aortofemoral bypasses, 30 axillary femoral bypasses, and 65 femoropopliteal bypasses (to but not beyond the knee joint). We have had opportunity to examine 20 of these grafts recovered at autopsy from 15 patients after implantation periods varying from 7 to 765 days. Only one of the deaths was graft-related, this being secondary to erosion of the right limb of an aorto-bilateral femoral graft into the third part of the duodenum.

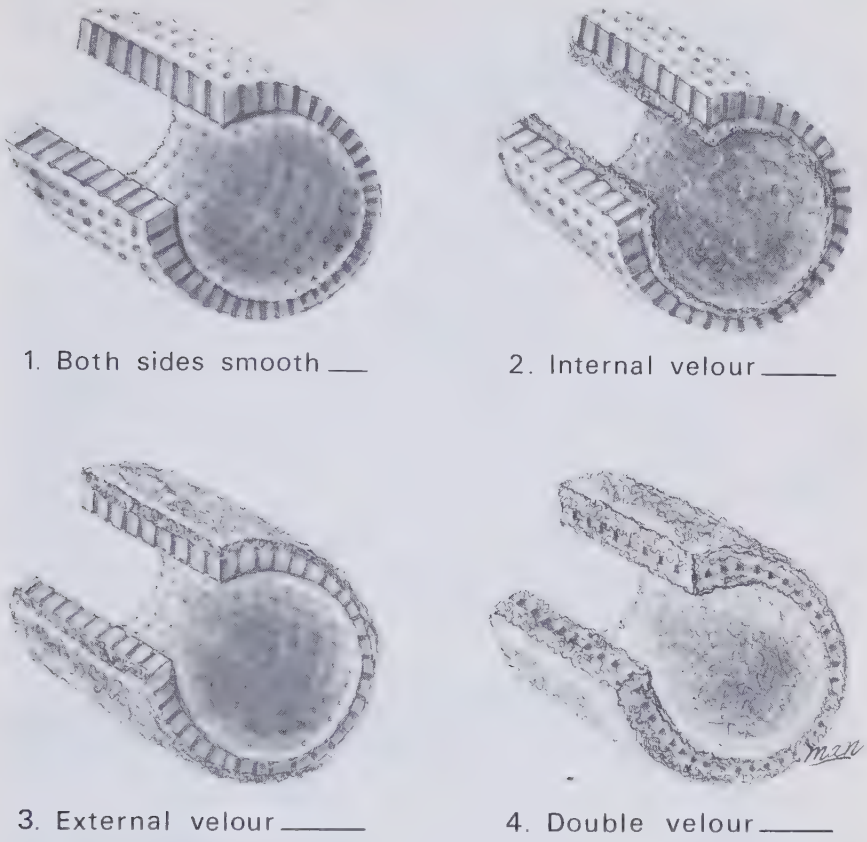

Figure 1. Diagrammatic views of the surfaces presented to the blood and perigraft tissues by conventional knitted prostheses with "smooth" walls and by knitted prostheses having velour surfaces inside, outside, or both.

These external velour specimens have been compared to 55 "smooth" walled knitted Dacron prostheses recovered at autopsy or reoperation from 40 patients after implantation periods ranging from 16 days to 11 years.[17] Histologic comparison showed that: (1) outer capsular adherence to the velour surface was very much greater than to the "smooth" surface of conventional knitted prostheses; (2) tissue ingrowth into the interstices of the velour prosthesis was enhanced by this adherence; and (3) organization of thrombus deposit on the inner wall was facilitated by the ingrowth (Fig. 2).

The outer capsular adherence to the external velour prostheses was significantly greater than that to conventional ones. In the latter, firm bonding does not take place until fibrous tissue has grown about halfway through the interstices, a process requiring about 3 months in man. The externally veloured graft is firmly anchored in about 1 month due to the entrapment of velour fibrils by fibrous tissue. (Bonding for both types of

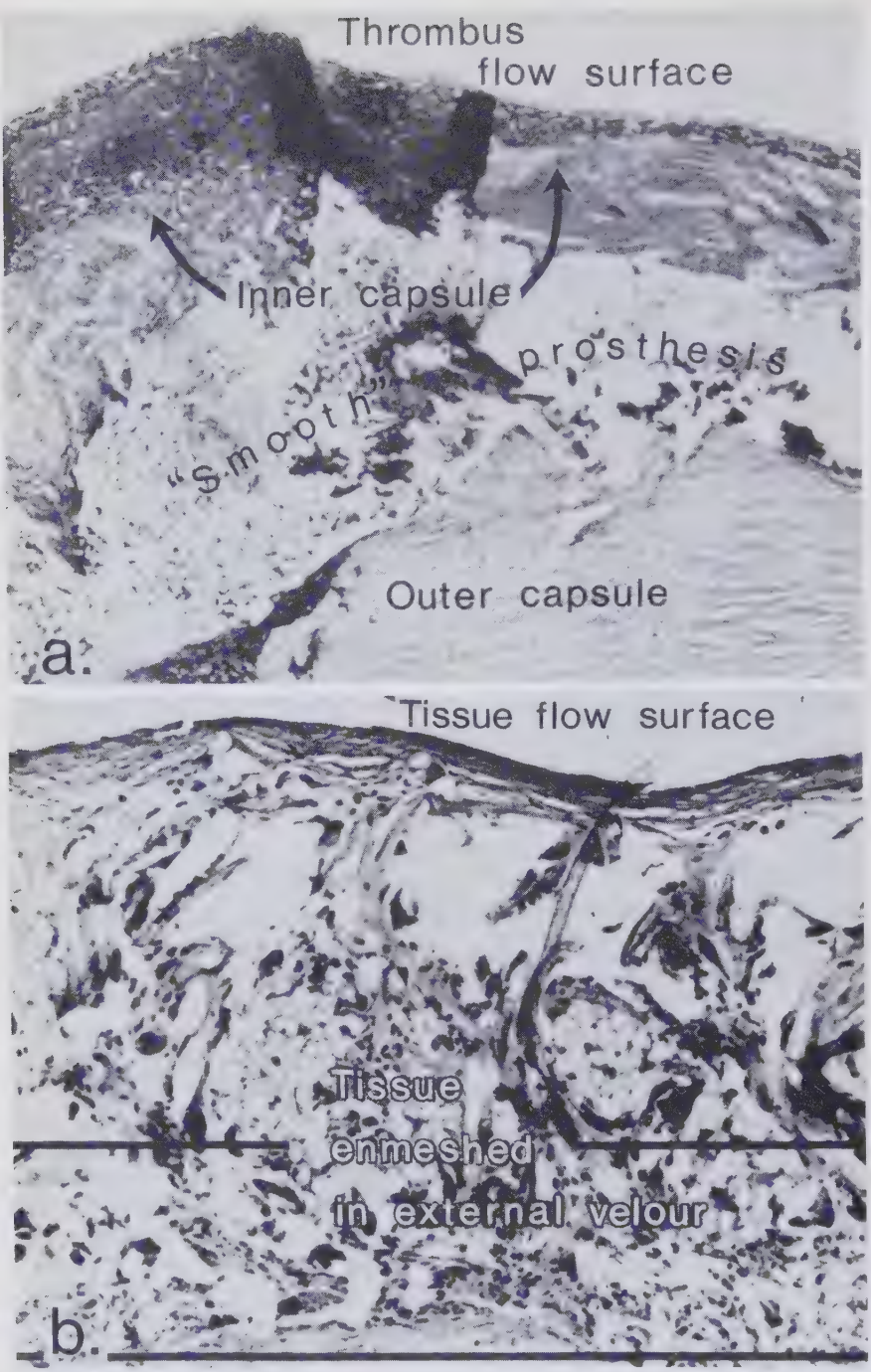

Figure 2. Comparative histologic appearance of midportion of aortofemoral grafts of (*a*) knitted Dacron prosthesis with "smooth" walls at 6½ years and, (*b*) knitted Dacron prosthesis with an external velour surface at 6 months. Note that the healing of the "smooth" walled prosthesis is incomplete at 6½ years – its flow surface is formed of compacted fibrin, a characteristic of this type of prosthesis. In contrast, the healing of the external velour prosthesis is much better, with a flow surface formed of thin, healthy fibrous tissue.

Figure 2 continued on opposite page.

grafts is markedly delayed by the presence of perigraft hematomas, seroma, infection, or a badly scarred tissue bed.)

Since the femoropopliteal location provides a severe test for prostheses because of low flow rates and angulation with motion, our results in this location are of interest. This series includes grafts to but not beyond the knee joint and includes only patients followed for 15 months or more. The series consists of 34 grafts. Of these, 21 (62 per cent) were either open at time of death (one graft at 18 months) or remain open in living patients (20 grafts from 16 to 35 months, average 24 months) without a thrombotic occlusive episode. Thirteen (38 per cent) of the grafts closed at an average of 10 months postoperatively (shortest 4 months and longest 24 months). Of these 13 grafts undergoing closure, seven were subjected to a thrombectomy procedure. Of these, six (86 per cent) remain open from 2 to 11 months (average, 8 months).

Thrombectomy was not done in six patients with closed grafts, either because their circulation was still adequate (three patients) or because in the early part of the series we did not think thrombectomy could be done for these patients. We had found with conventional prostheses that the poor bonding of the outer capsule to the prosthesis would not allow adequate Fogarty catheter balloon traction to be placed upon the prosthesis without dislodging it from its bed. Dislodging the graft caused it to become porous again as the fibrin mat was stripped away from the inner surface and the weak tissue attachments were stripped away from the outer wall. Later, we discovered that the secure bonding of the outer capsule to the external velour prosthesis enabled the procedure of thrombectomy to be done very simply through a small incision over the lower portion of the graft.

It is of interest that of the 34 grafts implanted, 27 (80 per cent) are now patent (26 grafts at an average of 24 months) or were so at the time of death (one patient at 18 months). This includes the six opened by thrombectomy. It is probable that this combined patency figure of 80 per cent for grafts never closing (21 of 34) and those opened by thrombectomy (6 of 7) would be higher had thrombectomy been attempted for the other six grafts that closed.

We have seen one case of complete healing (a bilateral aortofemoral graft at 178 days)[9] and three others with spotty complete healing throughout the graft, including one which was suitable for endothelial studies.[16] This specimen—a unilateral axillary to bilateral femoral prosthesis at 21 months—was available within an hour after death and showed endothelium wherever there was complete healing of the wall. In addition, an endothelial monolayer was also found atop extremely thin fibrin layers covering the underlying tissue bed in areas immediately adjacent to those completely healed.

We have concluded that our external velour prosthesis, while of definite value, is an incomplete application of the trellis concept. The superb

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Figure 2. *Continued.*

(Fig. 2*a* reproduced from Berger, K., et al.: Healing of Arterial Prostheses in Man: Its Incompleteness, Ann. Surg., *175*:118, 1972; used with permission of J. B. Lippincott Co. Fig. 2*b* reproduced from Sauvage, L. R., et al.: An External Velour Surface for Porous Arterial Prostheses, Surgery, *70*:940, 1971; used with permission of C. V. Mosby Co.)

3-year patency figure of 82 per cent reported by DeBakey[5] for femoropopliteal bypass grafts above the knee joint with a knitted internal velour prosthesis strongly suggests that a filamentous inner surface is also of value.

The development of a suitable double velour (veloured inner and outer surfaces) prosthesis may be of value in providing greater assistance to the ingrowing tissue in order to achieve complete healing of the prosthesis (Fig. 3). The additional advantages should be of special importance to grafts acting as conduits for low volumes of blood at slow velocities, especially in locations where the graft is subjected to distortion by body movement.

The filamentous character of the outer wall of a double velour prosthesis needs to be relatively coarse so as to facilitate attachment of the perigraft tissues, and to guide them into the interstices. The filamentous surface of the inner wall should be fine and serve both to anchor the initial thrombus layer and to promote rapid inner wall healing by guiding cellular ingrowth into the thrombus. If the total graft thickness can be kept to a minimum, the total amount of healing required by the perigraft tissues will be reduced and can be completed sooner.

The specifications we currently consider optimal for a lightweight, seamless double velour prosthesis are: outer velour height, 150 microns;

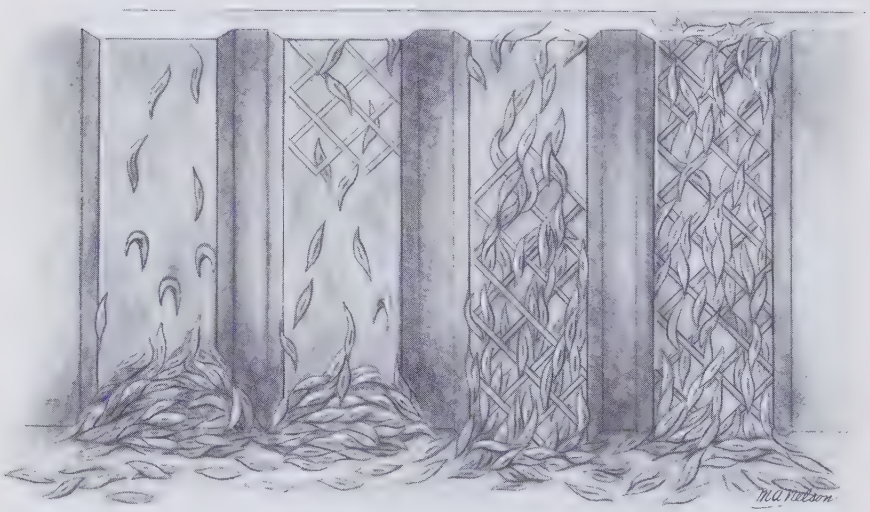


Figure 3. Conceptual view of the degree of assistance provided to the ingrowth of perigraft tissues by different types of arterial prostheses. In this schema, the degree of assistance is shown by the number of cells able to climb to the top of a wall which represents figuratively the flow surface of an arterial prosthesis. In (*a*), cells have difficulty reaching the top of the smooth wall. In (*b*), those cells reaching the upper wall receive assistance from the trellis provided by an internal velour surface. However, the cells have difficulty in reaching the trellis. In (*c*), the cells are greatly assisted in starting up the wall on the trellis provided by the external velour surface, but have no further help. In (*d*), cells receive maximal assistance in climbing to the top by a complete trellis—an arterial prosthesis with both external and internal velour surfaces.

graft body, 200 microns; inner velour height, 50 microns. The total thickness of this proposed prosthesis would be about 400 microns (0.4 mm) and it should have a water porosity of about 1500 ml per minute per $cm.^2$ This porosity should be adequate to allow tissue ingrowth and, at the same time, low enough to prevent significant implant bleeding. With United States Catheter Instruments, Inc.,* we are developing a double velour prosthesis according to these specifications.

Experimental and early clinical trial[11] of a prototype double velour prosthesis provides substantial support for the extended trellis concept. Nevertheless the basic disadvantage of pervious prostheses remains: there is a period of time, however greatly it is shortened by improved healing, when the graft is in danger of thrombotic closure. The ideal solution might be a temporary thrombus-resistant surface for protection during the healing period.

Trellis Construction with Coated Flow Surface. A distinction must be made between a wall that is impervious to blood but permeable to cellular ingrowth, and a wall with open interstices through which bleeding can occur. In routine clinical practice these interstices are closed by thrombus. Unfortunately, closure by thrombus renders the flow surface very thrombogenic. There would be great advantage if the interstices could be closed with a nonthrombogenic material that was at least as permeable to cellular ingrowth as thrombus.

Theoretically, anionic polyelectrolyte hydrogels appear advantageous as an alternative to thrombus as the ground substance. The hydrogel preparation has a negative charge (important for decreasing thrombogenicity) and is nonporous to blood but permeable to cellular ingrowth (vital to healing). We anticipate experimental trial of fabric prostheses with different types of surface coatings in the near future. From the drawing board standpoint (Fig. 4), this combination of the double velour trellis concept with a thrombus resistant nonporous flow surface permeable to cellular ingrowth is very attractive.

This solution to the problem of improving arterial prostheses, like the solution for a nonthrombogenic impervious wall, is in the future. Either solution might provide the desired characteristics for successful grafts in medium and small caliber arteries.

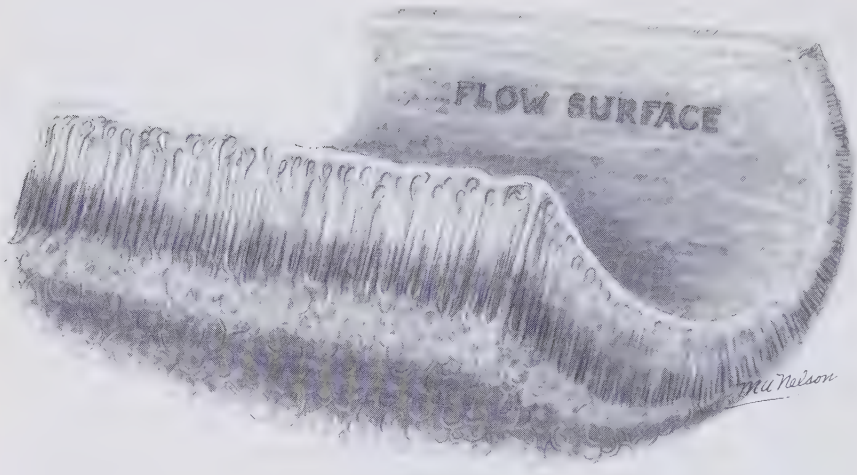
CONSIDERATIONS OF SPECIAL IMPORTANCE FOR PROSTHESES IN MEDIUM AND SMALL CALIBER ARTERIES

Factors important to prosthesis success in large arteries (aortoiliac) will apply even more strictly in medium-sized arteries (femoropopliteal) and are critical in small caliber arteries (coronaries and tibials). The following considerations may be of special importance to successful use of prostheses in small arteries.

Thrombotic Threshold Velocity

Synthetic grafts stay open as long as the velocity of blood flow through them exceeds that critical velocity allowing thrombus formation

*Billerica, Mass.



Double velour prosthesis with hydrogel flow surface

Figure 4. Diagrammatic view of double velour prosthesis with anionic polyelectrolyte hydrogel closing the interstices and forming the flow surface. In theory this compound prosthesis combines the completed trellis concept with a flow surface resistant to thrombus from time of implantation. It is hoped that such a design will provide a prosthesis in little danger of thrombotic closure even in low flow, small vessel environments.

to occur on the flow surface. We term this critical velocity the "thrombotic threshold velocity." As yet we have been unable to quantify this value for any graft. Once we are able to determine this value, it will be possible to compute a thrombotic threshold velocity index in terms of ml of blood flow per second per square centimeter of graft lumen area (ml/sec/cm^2). This index will serve as an important part of the pedigree of a prosthesis. Guided by this information, the surgeon would not implant a prosthesis in a flow environment where the velocity would be significantly below the thrombotic threshold of the prosthesis.

There may be value in conjecturing the series of events when a prosthesis is implanted in a flow environment of insufficient velocity. The blood reacts to the flow surface by layering it with thrombus until the lumen diameter is reduced to a point where the flow velocity rises just above the thrombotic threshold. The increased flow velocity prevents further thrombus formation, but the situation of velocity increase secondary to thrombus layering is highly undesirable in man. The additional thrombus probably prevents complete healing from ever occurring, as such a large amount of thrombus is too much for the ingrowing tissues to organize. As a consequence, the surface remains thrombogenic and lumen patency is maintained only through a tenuous equilibrium which can be unbalanced in favor of sudden thrombotic occlusion by even a modest drop in cardiac output.

The goal is to keep the inner thrombus layer very thin and to attain complete inner wall healing in the shortest time possible. With healing (including endothelialization), the thrombotic threshold velocity drops

far below the velocity of the flow environment. Such a favorable change provides significant protection against thrombotic closure.

Prevention of Perigraft Hematoma Formation

Anything that separates a graft from its surrounding tissues delays healing which, in turn, predisposes the graft to thrombotic closure. Thus, prevention of perigraft hematoma formation, important in any location, is of special importance in small arteries. This is particularly true for the coronaries, because hematoma formation in the mediastinum is frequent and difficult to detect.

The best protection against hematoma formation is and will remain meticulous surgical technique. An advantage of the yet undeveloped ideal impervious graft would be that hematoma formation would have no effect upon it.

Platelet Inhibition

Blood reacts to an artificial prosthesis by forming thrombus upon the flow surface. The surgeon can minimize this coagulation reaction in two ways: by avoiding an oversized prosthesis that will have a lower velocity than that of a more appropriate-sized prosthesis,[7] and by using anticoagulants to decrease the ability of the platelets to adhere to the flow surface.

We anticipate that agents which decrease platelet adhesiveness will be a necessary part of the postoperative program until full healing with endothelialization has occurred. The work of Harker and Slichter[6] and of Johnson[8] strongly suggests that arterial thrombosis is more a matter of platelet aggregation than it is of whole blood coagulation. Accordingly, the use of agents that decrease platelet adhesiveness (dipyridamole [Persantin] and aspirin) is advisable.

We have demonstrated[13] that the platelet survival time of patients receiving aortofemoral grafts, as well as of baboons receiving thoraco-abdominal grafts, is immediately reduced to about one-half normal (from 10 days to 5 for man, from 5 days to 2½ for the baboon). Persantin and aspirin return these survival times to normal. However, within a few days after these drugs have been discontinued, the platelet survival times revert again to their shortened levels.

In general, platelet survival in man returns to normal about 6 months after graft implantation; in the baboon it takes about 6 weeks. We do not know whether continuous treatment with agents that decrease platelet adhesiveness will shorten the healing time, but we believe that avoidance of platelet adherence to the flow surface will promote better, faster healing. Accordingly, our current policy is to keep all patients receiving arterial prostheses in appreciable risk areas such as the relatively low flow femoropopliteal location on Persantin and aspirin for 6 months with an additional month to taper off usage of these drugs.

SUMMARY

Conventional prostheses—pervious knitted tubes with "smooth" surfaces—are inadequate for use in small arteries and, for the most part, are

far from ideal in large ones due to the persistent high thrombogenicity of the flow surface, a consequence of the incomplete healing which characterizes these grafts. The thrombogenic nature of the flow surface of these pervious prostheses is greatly increased when the interstices are closed by thrombus at implantation, an essential reaction to stop bleeding. Furthermore, this initial thrombus layer continues to attract more thrombus until the process is halted by a sufficiently high velocity of blood flow. Complete healing of the entire wall, including establishment of an intact endothelial covering for the flow surface, would lower the thrombogenicity of the flow surface to essentially that of a normal artery. But this does not occur with conventional prostheses, as in man, unfortunately, this type of prosthesis does not heal completely even with many years' implantation.

Thus there is a need for new approaches in the development of arterial prostheses for use in small caliber arteries. The healing of smooth-walled porous prostheses is too slow. To develop prostheses that will be suitable for small vessel use two options are available: (1) eliminate the necessity for healing, or (2) aid and speed the healing process to completion. An impervious prosthesis whose flow surface was thrombus-resistant would be ideal. Such a prosthesis would not bleed at implant nor thrombose later. Healing through the wall could not occur, but there would be no need for it. Unfortunately, no suitable material has thus far been developed with which to make such an ideal prosthesis. All materials tested to date are much more thrombogenic than the intimal surface of a normal artery or of a completely healed pervious fabric prosthesis.

The other option is construction of a pervious prosthesis whose design favors healing by serving as a trellis for transinterstitial ingrowth of aerolar tissue. Grafts with "rough" or filamentous surfaces (outer or inner) are already in use. Clinical results with an external velour design indicate that healing and outer capsule adherence are greatly superior to standard "smooth" porous prostheses. But healing is still not as rapid nor as complete as desired in most cases, and therefore an effort is being made to extend the concept to both surfaces. Since we have shown an *external* velour surface to be valuable, and DeBakey has shown an *internal* velour surface to have merit, we are developing a "double velour" prosthesis in cooperation with USCI. This extends the trellis concept of graft construction to the entire wall in an effort to further accelerate healing.

However, unlike the ideal impervious graft—whose optimal characteristics would be present from implantation—any pervious graft has to remain patent through the dangerous period from implantation to complete healing of the flow surface. A possible solution to the problem of high thrombogenicity of the flow surface would be a graft with interstices sealed by a nonthrombogenic material which was permeable to cellular ingrowth. The danger of lumen closure by thrombus in low flow environments would be significantly lowered, protecting the graft until the flow surface is completely healed. Anionic polyelectrolyte hydrogels are under study as a means to seal the interstices and line the flow surface of veloured fabric prostheses. The challenge at hand is to develop a prosthesis that is no longer subject to closure except under very low flow

states. Such a prosthesis would be a major improvement for large caliber arteries and a necessity for small caliber ones.

There are some general considerations important to the use of prostheses in large vessels (aorta and iliacs) that will probably apply even more strictly when effective grafts are developed for medium (as popliteals) and small (as coronaries and tibials) caliber arteries. One of the keys to patency relates to velocity of blood flow through the graft, a concept which this paper refers to as "thrombotic threshold velocity" and which we are trying to quantify (ml/sec/cm^2 of graft lumen area) as a guide to the surgeon in selecting proper prosthesis size. Healing of small caliber prostheses will be effectively delayed in the presence of perigraft hematomas. This may be a particularly difficult problem to prevent in the case of aortocoronary bypass grafts. A final consideration is platelet inhibition. Administration of drugs such as Persantin and aspirin impedes platelet adhesiveness. Postoperative use of such agents appears to be of value until healing of a porous graft is completed and is recommended for prostheses implanted in low flow environments.

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Current Thoughts on Venous Thromboembolism

*N. L. Browse, M.D., F.R.C.S.**

The past 10 years have seen many articles written about the problems of deep vein thrombosis and pulmonary embolism. Most of these have been stimulated by new techniques of diagnosis which have enabled the disease to be studied in a scientific manner during life. In spite of all this work many people still die of pulmonary embolism and a visit to any hospital ward will quickly show that we are a long way from solving the problems of this disease. This paper will review the general situation and discuss the unsolved problems rather than relate the basic details of the newer techniques. Concepts are continually changing and past publications should be reviewed with caution. As John Hunter said, "Do not ask what I have written or said, but what I think." The personal opinions which this paper contains are based on facts as they are currently known, or believed to be known, in March 1973, but they will be different tomorrow and probably invalid.

THE DISEASE

In the past few years our attitude towards thromboembolism has changed. It was once thought to be a relatively uncommon cause of sudden death, but we now appreciate that it exists in the majority of our patients. There has been no real change in the disease; the pathology books of the end of the nineteenth century and many more recent autopsy studies all stress the high incidence of deep vein thrombosis and pulmonary embolism in patients who are clinically normal. Clinicians have been slow to appreciate this point, but the advent of the fibrinogen uptake test (F U.T.), which has enabled us to diagnose small clinically silent thrombosis, has made everyone acutely aware of the ubiquity of this disease. We no longer think of a few patients dying from pulmonary embolism, but that one-third of the patients in every hospital have thrombi in their calves.

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We see three forms of venous thromboembolism today, the variety detected by the F.U.T., the clinically detectable deep vein thrombosis, and pulmonary embolism. Each of these may be present alone or in combination with the others.

The ^{125}I-Fibrinogen Detectable Thrombus

Between 1967 and 1968 studies at King's College Hospital and the Middlesex Hospital revealed that approximately 30 per cent of patients develop calf vein thrombosis after a surgical operation. Since then others have shown that a similar proportion of patients get thrombosis after myocardial infarction and other serious medical illnesses. Unfortunately the F U T. examines the leg only from the mid-thigh to the ankle and many studies have shown that large thrombi can begin in the iliac, common femoral and profunda femoris veins, so it is an incomplete examination. Nevertheless, at least 80 per cent of all thrombi begin in the calf and a small proportion of these grow and extend above the level of the knee into the popliteal and superficial femoral veins. All these are detected by the F.U.T. In the King's College series the proportion that extended was 10 per cent; in my own series it is approximately 2 per cent.

It is claimed that thrombosis in the calf does not give rise to serious embolism and that there is a threat to the patient's life only when extension occurs, but the size of thrombus that can come from the calf depends on the size of the calf veins and, in one particularly large vein, we measured a thrombus 12 cm long and 0.75 cm in diameter. Such an embolus might not kill a fit person, but it could be fatal in someone with a sick heart or previous embolism.

Failure to appreciate the limitations of the F.U.T. has even caused some to claim that pulmonary embolism does not occur in the absence of thrombus in the calf. We have seen many patients with thrombosis beginning in the femoral and iliac veins without calf thrombosis. The concept that thrombosis always starts in the calf, and that pulmonary embolism occurs only when the thrombosis propagates into the superficial femoral vein, is not true. We do not know the exact proportion of F.U.T. detectable peripheral thrombi that propagate, or how many emboli come from this type of thrombus compared with the more proximal thrombi, and we will never know if we confine our attention to the lower leg and the F.U.T.

Why do so many patients get small thrombi in the calf? Haematologists postulate that there is a delicate balance between coagulation and fibrinolysis. We do not understand which factors affect this balance, if indeed it exists, but the fact that so many people get small thrombi, which usually disappear spontaneously, seems to support the hypothesis. Perhaps an operation or illness tips the balance transiently towards thrombosis, causing small thrombi to develop in the calf, but when the balance swings back the thrombi stop growing and then lyse spontaneously. If this suggestion is true then many factors may alter the balance. Alterations in the coagulability of the blood are likely to be just as important as stasis, and the cause of venous thrombosis may differ from patient to patient.

The factors that alter the coagulation/lysis balance and initiate the

thrombosis may not be the same as those that cause the thrombus to grow, and procedures that prevent the initial thrombus may not prevent a thrombus growing. There is still a great deal to be learnt about the aetiology of venous thrombosis and although it is a useful research tool, the F U T. has told us little about the underlying cause of the disease. It has simply confirmed that the interpretation of the autopsy findings of the past 100 years is relevant and applicable to the patient during life.

Clinically Detectable Deep Vein Thrombosis

Clinically detectable deep vein thrombosis is present in 5 to 10 per cent of patients. Comparing this figure with the incidence of thrombosis detected with the F.U.T. (20 to 30 per cent) shows that physical signs are an unreliable method of detection, but this does not mean that they have no value. Signs that indicate obstruction to the venous outflow from the leg – oedema and distention of the veins – are reliable but pain and tenderness are not. In some studies nearly 50 per cent of the patients with calf tenderness did not have thrombosis. This raises the question, what causes the pain of venous thrombosis? Two patients may have identical phlebograms, but one may have no physical signs and the other marked tenderness. Is the presence of symptoms related to the aetiology of the disease, the type of thrombus or the properties of the vein wall? There is no published long-term follow-up of the state of the deep veins 5 or 10 years after the silent thrombosis detected with the F.U.T., but very few of the 300 patients that I have seen with this diagnosis have yet developed any physical signs which might be attributed to it. On the other hand it is my impression that a greater proportion of those who had pain and tenderness have developed signs of valvular incompetence. It could be argued that the presence of physical signs is just a feature of the extent of the thrombosis, but this is probably not the answer and it may be shown that the factors responsible for the presence or absence of physical signs affect the late sequelae.

Pulmonary Embolism

We know nothing about the mechanisms that make thrombi fragment and embolise. Radiographically identical thrombi can behave in dissimilar ways – one will break up and embolise and the other will retract and become fixed without fragmenting. There are two likely causes of fragmentation, mechanical stress and intrinsic dissolution. Many think that mechanical forces are the main cause and use the fact that pulmonary embolism often occurs at the same time as defaecation to support their argument. But emboli also occur when a patient is lying quite still, even during anaesthesia, and so I suspect that the most important factor is the changes that are occurring within the thrombus. The measurable constituents of thrombus vary only slightly from patient to patient, but occasionally a thrombus kept in vitro will dissolve spontaneously or be totally resistant to lysis, so there must be differences. A test which would tell us which thrombi are undergoing autolysis and will fragment and embolise would be an extremely valuable aid to clinical management.

DIAGNOSIS

In the previous section a number of diagnostic tests have been mentioned in connection with their contribution towards our understanding of the disease, but their value at the bedside is worthy of separate discussion. No treatment should be given if the diagnosis is based solely on physical signs, unless they are so gross that an incorrect diagnosis is impossible, but any clinical suspicion must be investigated.

The Ultrasound Flow Detector

The introduction of the ultrasound flow detector has been an extremely valuable advance in clinical management. With this apparatus it is possible to detect flow in the main veins above the knee and when it is present it is reasonable to assume that the veins do not contain a massive thrombus. The technique is not 100 per cent accurate because small thrombi usually lie against the side wall of the vein and do not obstruct the flow of blood, but once they come to occupy more than 50 per cent of the cross-sectional area of the vein they will produce sufficient obstruction to be detectable. Small veins such as those in the calf, thigh and buttock muscles cannot be studied. Provided the clinician is aware of these deficiencies the technique is extremely useful. The state of the major veins of the limbs can be assessed in any patient, however ill, in 2 to 3 minutes. A positive test should be taken as an indication for precise anatomical diagnosis by phlebography. A negative test indicates that a major embolus is most unlikely.

Plethysmography

The rate at which blood flows through the veins as it leaves the leg after venous occlusion is determined by the resistance of the veins. If the veins contain thrombus their resistance will be increased and the emptying of the calf after congestion prolonged. This principle has been used in the various methods of plethysmography that have been advocated. It is basically the same principle as that used by the ultrasound method and is no more accurate, but can be used on the calf. It is a little more difficult to apply and has no outstanding advantages.

The Fibrinogen Uptake Test

Some of the results of clinical research using this test have already been mentioned. For the clinician, this test has a number of serious practical disadvantages. The fibrinogen must be obtained from hepatitis-free donors, and at present there is no commercially available fibrinogen approved by the F.D.A. The test is costly, in England one dose of ^{125}I-fibrinogen costs approximately $8, and one technician can study only 25 patients each day. It is unsuitable for emergency situations because the patient should be prepared with oral potassium iodide to block the uptake by the thyroid gland of any free iodine for at least 24 hours, although this can be omitted in an emergency, and after giving the fibrinogen it is necessary to wait 24 hours until there is sufficient radioactivity in the thrombus to make it detectable. Most important of all, it is only possible to

examine the calf and the middle and lower third of the thigh, so it is an incomplete examination of the deep veins.

I have found the F.U.T. useful for clinical research and for the diagnosis of the very common problem of pain and tenderness in the calf. Small thrombi in a muscle vein can be missed by phlebography, and in these circumstances the F.U.T. may be more accurate. Provided the thrombus is less than 5 to 7 days old it will take up sufficient fibrinogen to make it detectable, even if the patient is anticoagulated, but when the thrombus is more than 7 days old or if the patient has been on anticoagulants for more than 5 days the thrombus will not take up the fibrinogen and there is a 40 to 50 per cent incidence of false negatives.

The great advantage of the F.U.T. is the facility to do daily examinations and monitor the progress of the thrombus for 5 to 7 days. When a thrombus is detected it need not be treated unless subsequent examinations show that it is enlarging. A positive or negative F.U.T. gives no indication of the likelihood of pulmonary embolism.

In view of its cost the F.U.T. is unlikely to be used to screen all patients, but I find it very helpful for screening high-risk patients, particularly if it is combined with frequent examinations with the ultrasound flow detector.

Phlebography

Phlebography is the most accurate and complete method of diagnosis that we have. In addition to giving a diagnosis it provides information on the exact site, extent and nature of the thrombus. Phlebography is not a perfect examination, for it only displays the profunda vein and its tributaries in 40 per cent of cases, never shows the tributaries of the internal iliac vein and can never show every vein within the calf, but the veins which are likely to produce large emboli can always be displayed. Phlebography can be done in any hospital that has an x-ray table, as it requires no more than the injection of a radiopaque dye into a foot vein. Image intensification and television display improve the quality of the films but are not essential. Occasionally the injection is painful and a few patients develop a superficial thrombophlebitis. The radiologist must ensure that all the veins are quickly emptied of contrast medium after the examination, by exercising the leg and flushing with heparinised saline.

The interpretation of phlebograms is straightforward. After a little practice it is easy to recognize the causes of false positive reports such as knot-holes from the entry of side branches, streaming and extramural impressions. There is no published study which compares the reliability of phlebography with postmortem examination, but as far as the major veins are concerned all agree that it is an extremely reliable investigation. Thrombi removed surgically usually fit exactly over their phlebographic shadow.

Clinical Application of Diagnostic Techniques

The above methods of diagnosis can be used in a variety of ways. When a patient has a pulmonary embolism it is mandatory to order bilateral phlebograms. So many small pulmonary emboli herald a second

embolus that the state of the deep veins must be assessed in order to treat any residual thrombus. If for some reason phlebography is not possible, then the veins can be quickly examined with the ultrasound flow detector to see if there is any major vein occlusion.

The physical signs of major deep vein thrombosis, such as a white or blue leg, are also an indication for phlebography because detailed information about the thrombus is needed before deciding on treatment.

The majority of patients, however, present with a few minor physical signs in the legs and it is impractical to perform phlebograms on them all. For these patients a screening examination with the ultrasound flow detector followed by the F.U.T. is the simplest method of assessment. If the ultrasound examination is positive they must have a phlebogram. If it is negative then it is safe to wait 24 hours for the result of the F.U.T. before beginning treatment.

It is apparent that our diagnostic methods are far from ideal. We do not have a simple noninvasive bedside technique that can be applied to all patients, which will identify the presence of thrombosis in any vein and at the same time give an indication of the age of the thrombus and its fixity to the vein wall. Whether we will ever have such a method remains to be seen but there is no reason to be complacent with our current methods.

TREATMENT

In most cases our treatment of deep vein thrombosis is grossly inadequate in that we do little more than relieve the symptoms and stop further thrombosis. Although this may prevent some pulmonary emboli it does not help the veins and in the long term leaves them damaged and inadequate. The ideal treatment of deep vein thrombosis should remove all the thrombus from the veins and prevent further thrombosis, so that pulmonary embolism cannot occur and the valves still function. Theoretically this might be achieved by surgical or pharmacological methods.

Thrombolysis

We have two drugs which are capable of dissolving thrombus in vivo: streptokinase and urokinase. Both are enzymes which activate the fibrinolytic system, converting plasminogen to plasmin which in turn dissolves fibrin to fibrin breakdown products. It is believed that streptokinase works by entering the thrombus and activating the plasminogen contained therein. This is an important concept because it draws attention to the fact that thrombi are not inert static areas in the bloodstream. When radioactive fibrinogen is given to a patient with established thrombus it passes into the thrombus and in one of my own cases the radioactivity within a thrombus, known to be at least 3 weeks old when removed at autopsy, was four times higher than that of the peripheral blood 2 days after the injection of the fibrinogen. After a nonadherent thrombus is dissolved the valves may be left intact, but if the thrombus is adherent to the

vein wall the valves will be inflamed and thickened and will become incompetent in spite of successful thrombolysis.

The side effects of thrombolytic drugs can be serious– bleeding, pyrexia, allergic reactions–but they can usually be controlled or reversed. In practice I have found that the only reason to stop treatment is bleeding. Arteriotomies are particularly prone to bleed, and I will not use streptokinase within 5 days of any form of vascular operation.

The disadvantage of streptokinase and urokinase is that they work through the natural fibrinolytic system which has its own inbuilt system of inhibitors. In the future I expect that progress in thrombolysis will be with drugs that act directly on fibrin. There are such drugs available, but at the moment they are not safe to give to man because they dissolve the whole patient rather than the thrombus, but this is a field worthy of further study.

Thrombectomy

Surgical thrombectomy has been practised since the 1930's and in certain circumstances is a very quick and efficient way of removing thrombus. It is most effective with fresh nonadherent thrombus, the same type of thrombus that can be dissolved by streptokinase. Adherent thrombus can be removed, but some is inevitably left on the wall of the vein and the chance of re-thrombosis is high. The surgical technique became simple and available to all with the development of the Fogarty balloon catheter. Many people had tried many methods of thrombectomy, including catheters with balloons, but Fogarty was the first to appreciate that a fine catheter could be pushed through a thrombus without causing it to break up or detach from the vessel wall so that its balloon could be blown up when the tip had passed beyond the end of the thrombus.

The difficulties of thrombectomy are that it is sometimes difficult to pass the balloon catheter peripherally through the valves, and the small tributaries are beyond reach. Its main asset is that it is a simple operation that can be done under local anaesthetic. A patient can be protected from embolism in less than an hour, in contrast to the 6 hours taken by streptokinase. It is tempting to combine thrombolysis with surgical thrombectomy, but it can be hazardous. If full body doses of streptokinase are given there is bleeding from the venotomy, and if smaller doses are given locally there is insufficient thrombolysis.

Prevention of Embolism

Pulmonary embolism will stop when we can prevent deep vein thrombosis, but in this section I wish to deal with the prevention of embolism from established thrombosis. A considerable proportion of patients have a herald embolus before their massive embolus. Investigations of the limbs by bilateral ascending phlebography, supplemented if necessary by perfemoral or intraosseous injections, are essential if a patient has had any physical signs suggestive of pulmonary embolism. With the extent of the residual thrombus delineated, a plan of treatment can be made to prevent further embolism with minimum damage to the veins. Thrombolysis or thrombectomy can be used to remove fresh thrombus in

large veins, but when there are multiple small peripheral thrombi it is safer to ligate the vein upstream and confine the thrombus to the limb. The vogue in America has been to obstruct the vena cava. The cava has been ligated, plicated, clipped, sieved, umbrella'd and stenosed. This is usually unnecessary, as in most cases the thrombus is confined to the limbs and ligation of a more peripheral vein is adequate treatment. In my own experience, ligation of the superficial femoral vein below the entry of the profunda vein has been sufficient for more than 80 per cent of cases.

There is always argument about the morbidity of these operations. Everyone has seen gross oedema of the limbs following caval ligation, but it is not very common and is not just due to the surgical procedure. It depends on the degree of thrombosis present in the vein before the operation and the ability of the patient to develop a collateral circulation. Swelling can be minimised with high elevation and good elastic support. I have not found ligation of the superficial femoral vein to cause any short-term disability. In two groups of patients with similar forms of thrombosis, half with a superficial femoral vein ligation and half treated with anticoagulants alone, reexamined 3 to 5 years later, there was no difference in the incidence of swelling in the leg, nor in the incidence of clinically detectable varicose veins.

Anticoagulants

In most cases the residual thrombus after an embolus is small and anticoagulation would be sufficient treatment, but we do not have an ideal anticoagulant. Heparin must be given intravenously or subcutaneously and is not suitable for prolonged use, and oral anticoagulants are affected by so many factors that they can be exceedingly difficult to control. Nevertheless, we can usually prevent further thrombosis with these drugs and give the existing thrombus a chance to retract and organise.

Anticoagulants have no direct effect on existing thrombosis. Heparin does not prevent thrombus fragmentation and embolism. The reduction of the mortality rate after pulmonary embolism by heparin is due to its ability to stop new thrombus forming around the embolus and in the limbs. This is why treating pulmonary embolism with heparin alone is not adequate. The peripheral veins must be examined and treated.

Defibrination by snake venoms such as arvin and reptilase is a very effective method of anticoagulation, but is not yet commercially available. It has the great advantage of producing an easily measurable effect because once there is no fibrinogen to measure in the blood the patient cannot coagulate. I expect that this form of anticoagulation will be used more often over the next few years, especially if the drug can be produced in a form that can be given subcutaneously, rather than intravenously, without stimulating antibody production.

PULMONARY EMBOLISM

The most important advance in the diagnosis of pulmonary embolism in the last 10 years has been the development of the lung scan. Provided

the other causes of perfusion defects are appreciated, and if the scan is always considered in conjunction with the clinical signs and the chest x-ray, it can often replace the pulmonary angiogram. However, pulmonary angiography, like phlebography, is the ultimate anatomical method of diagnosis and is essential before performing an embolectomy, unless circumstances are so acute there is no time for it. Angiography is often said to be a hazardous procedure. This is because many fail to appreciate that most anaesthetic and radiopaque dyes are strong vasodilators, and their effect must be countered by vasoconstrictor drugs given before the patient is anaesthetised.

The techniques of pulmonary embolectomy are well established. As pulmonary embolectomy under cardiopulmonary bypass can be performed only at special centres, the development of embolectomy during caval occlusion has been a useful advance, for it is an operation that can be performed by any competent surgeon. The sternum is split and the pericardium opened. The carvae are snared and intermittently occluded for periods of 3 minutes, while the pulmonary artery is opened, sucked clean and then side-clamped to allow the caval clamps to be released. The caval occlusion reduces the blood loss from the pulmonary artery to reasonable levels. Patients died from the old Trendelenburg operation because there was no method of controlling the blood loss once the embolus was sucked out.

Streptokinase and urokinase can dissolve fresh pulmonary emboli. The problem with embolectomy and thrombolysis is to decide when they are required, for it is well known that the majority of patients who do not die instantly from their pulmonary embolism get better and are left with few or no long-term sequelae. At the moment surgical embolectomy is reserved for those who are rapidly deteriorating or who are being kept alive by external cardiac massage. The remaining patients can be divided into those who slowly deteriorate, or are extremely ill and show no signs of improvement, and those who, having collapsed, steadily improve. The latter group are the majority and should be managed with heparin and investigation and treatment of their limbs to prevent further emboli. The former group are presently treated with streptokinase. Patients given streptokinase have a greater reduction of their pulmonary artery resistance, compared with those treated with heparin after 12 and 24 hours of treatment, but by 7 or 8 days the difference is not so marked. In the few patients studied at 6 months there is no difference. Thus, streptokinase need only be given if the clinician believes that the pulmonary artery obstruction is endangering the patient's life or may cause long-term sequelae. This is a very difficult assessment.

THE PREVENTION OF PULMONARY EMBOLISM

Recently journals have been crowded with papers on the prophylaxis of pulmonary embolism. These studies have been made possible by the advent of the F.U.T. Many techniques will reduce the incidence of the calf thrombi detected by the F.U.T., but there is no evidence that any of

them affect the incidence of pulmonary embolism. It is assumed that if a method reduces the incidence of thrombosis in the calf it will reduce the incidence of pulmonary embolism. This has yet to be proved. Methods such as electrical stimulation of the calf, pneumatic stockings, pedal machines, anti-platelet substances and subcutaneous heparin must remain subjudice until their effect on embolism is established. Until then the individual clinician must decide whether the effect any method may have on pulmonary embolism outweighs the possible harm it might do. Both are unknown quantities. There is a real possibility that we will have an effective form of prophylaxis for surgical patients within the next few years. The situation is so fluid at this moment that there is no point in enumerating current studies.

Prophylaxis for medical patients is more difficult, because these patients do not reach us until after their illness has started and the thrombosis has usually begun. We must remain alert and try to prevent embolism by making an early diagnosis. This can be done by screening with the ultrasound flowmeter and the fibrinogen uptake test, followed by the administration of anticoagulants if there is any sign of thrombosis above the knee or if the small common calf thrombus begins to enlarge. Far more good will be done at this moment in time by vigorous investigation and treatment, than in the application of unproved forms of prophylaxis.

I hope it is apparent from this paper that although we now have methods of diagnosis and treatment which allow us to see and sometimes confine this enemy, we have not mastered it and we must increase our efforts to improve our understanding of the aetiology, diagnosis and treatment of this troublesome disease.

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Surgical Treatment of Cerebral Ischemia

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The significant role that cerebral arterial disease plays in the mortality and morbidity of the general population, particularly in those with advanced age is well known. In the United States stroke ranks third as a cause of death.[1] Approximately 1½ million persons are afflicted by stroke annually. When considering the loss of life and disabling physical and mental sequelae resulting from cerebrovascular accident, its economic significance becomes quite clear. It has been estimated that stroke costs this nation more than one billion dollars yearly.

Many diseases affect the cerebral vessels, but atherosclerosis is the underlying cause for the majority of strokes. A remarkable change in the concept of pathogenesis of cerebral infarction has occurred during the last quarter of a century. The advent of cerebral angiography and the development of techniques of reconstructive vascular surgery have demonstrated beyond doubt that extracranial occlusive disease rather than intracranial involvement is the major culprit in producing ischemic infarcts or embolic lesions. There is a significant incidence of extracranial occlusive disease occurring in the general population as well as in the group of individuals already exhibiting symptoms of cerebrovascular disease. Approximately 15 per cent of individuals seen in our service for other vascular lesions exhibit findings of occlusive disease in the brachiocephalic system.

Historically, development of knowledge of the correlation between cerebral dysfunction and extracranial arterial lesions susceptible to surgical treatment developed in two rather distinct anatomical areas. The clinical features associated with obstructive lesions of the aortic arch branches were appreciated first by Broadbent in 1875.[2] He described arteriosclerotic occlusion of the proximal portion of these branches and

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noted their segmental nature. His interest in the patient presented was primarily that of explaining the bilateral absence of radial pulsations in the presence of normal pulses in the lower extremities. Hunt, in 1914,[13] re-emphasized the importance of occlusion of the aortic arch branches in the production of cerebral symptoms.

Ophthalmologists were the first to describe the relationship of obstructive lesions of the brachiocephalic branches to visual disturbances. Takayasu[27] of Japan wrote of a 21-year-old woman who was under treatment for inexplicable early cataracts. She had no pulses in her upper extremities or neck. Thereafter, other ophthalmologists reported such cases, mostly occurring in young women. Many of these authors were Japanese, and finally Shimizu, in 1948,[26] in reporting six additional cases, called the condition "pulseless disease." In 1952, Caccamise and Whitman[3] called the condition "Takayasu disease." The first description of a syndrome caused by occlusion of the aortic arch branches which did not necessarily emphasize the ophthalmic manifestations was reported by Martorell and Fabre[19] in 1944. Cerebral and upper extremity symptoms were common in their series. Martorell emphasized that the mechanical obstruction in the proximal end of the brachiocephalic branches was due to atherosclerotic involvement, unlike the reported cases by the Japanese group which were caused by a nonspecific arteritis. Clinical manifestations of cerebral ischemia in these cases were basically blamed on diminished forward flow until 1960, when Contorni[5] observed retrograde flow in a patient with arteriosclerotic obstruction of the subclavian artery. Reivich and co-workers, in 1961,[24] emphasized that in addition to decreased forward flow a reversal of flow from the brain to supply the arm circulation occurs when the obstructive lesion is situated strategically in the subclavian artery proximal to the vertebral subclavian junction (subclavian steal syndrome). The importance of restoration of flow to the obstructed subclavian artery in patients with cerebral symptoms thus became clear.

The second area of endeavor was related to the obstructive lesions of the cervical portion of the arteries leading to the brain, the lesions here occurring principally at the common carotid bifurcation and at the origin of the vertebral artery. Chiari, in 1906,[4] and Hunt, in 1914,[13] stressed the importance of examining the cervical portion of the carotid artery at autopsy of patients dying of stroke. Moniz and co-workers[20] in 1937 succeeded in demonstrating the occlusive lesion of the cervical portion of the carotid artery in living patients following cerebral angiography. Interest in reconstructive vascular surgery in the brachiocephalic system began, however, when Eastcot, Pickering and Rob[7] in 1954 described reconstruction of the internal carotid artery in a patient with intermittent attacks of hemiplegia. This report heralded a new era in the surgical management of cerebral vascular disease.

Our interest in this field began in 1956,[6] when a patient with complete occlusion of the innominate and left subclavian arteries was subjected to an endarterectomy of the innominate artery. The endarterectomized segment later thrombosed, requiring an aorto-carotid bypass graft. Our first experience with management of carotid bifurcation dis-

ease was in December 1955, when a patient with transient stroke syndrome was operated upon for an obstructive lesion of the origin of the internal carotid artery.[18] The occluded segment was excised and a segment of the long saphenous vein was employed to bridge the defect. This patient had total relief of cerebral symptoms following surgery. Since these early exposures to these lesions, 1400 patients have been evaluated during the last two decades, and the present report summarizes our current concept of the role of surgery in the management of the extracranial occlusive disease of the brachiocephalic system in prevention of stroke and in the palliation of cerebral symptoms.

ARTERIOGRAPHY

The Department of Neuroradiology is solely responsible for visualization of the brachiocephalic system in our institution. The indication for angiography is based on the patient's symptoms and clinical findings. We have advocated carotid artery visualization in all patients with carotid bifurcation bruit, regardless of the presence or severity of their symptoms. The justification for this approach is that in the last 2 years, in a series of about 300 patients yearly, there has been no incidence of death following angiography and there have been only two instances of unilateral neurological deficit following carotid visualization. One of these patients developed thrombosis of the common carotid artery and the other lost a carotid bifurcation bruit following the percutaneous carotid arteriogram. Both patients were treated as an emergency with total restoration of flow and full recovery. Minor complications such as local hematoma and subintimal injections of the artery or allergic reactions were few. None resulted in permanent sequelae. Bilateral percutaneous carotid arteriogram has remained as our first priority in patients with clinical evidence of carotid bifurcation disease. In those patients with a history of vertigo, ataxia or bilateral visual difficulties, a right brachial blowback injection in addition to a left percutaneous carotid arteriogram would often give the pertinent information. Visualization of the vertebral basilar system is often possible by this approach if the right vertebral artery is patent. Selective brachiocephalic angiography via the femoral artery is occasionally indicated for visualization of the proximal lesions in the branches of the aortic arch.

ASYMPTOMATIC INTERNAL CAROTID STENOSIS

Since we adopted the policy of routine angiographic study of all patients with clinical evidence of carotid bifurcation disease prior to recommending other major surgical procedures, we observed that many asymptomatic patients have severe stenotic lesions at the origin of the internal carotid artery. In two instances where highly stenotic lesions were ignored, the patients had to be operated upon as an emergency when the carotid bruit disappeared and a neurological deficit developed on the appro-

priate side. Some of the patients in this group who did not develop cerebral manifestations while in the hospital did experiefce stroke months or years later. This observation indicated to us the value of carotid endarterectomy in asymptomatic patients prior to subjecting them to other vascular procedures.

Our conviction became even stronger when we studied the natural history of carotid atheroma[14] in 140 patients with mild to moderate involvement of the carotid bifurcation. A long-term study of these patients revealed that 11 died of stroke during the interval, seven died of cardiac causes, and another seven died of unrelated causes. Ninety-three patients had repeat angiography. Significant changes had taken place in 51 patients, 22 of whom required carotid endarterectomy. The rate of atheromatous change appeared to be directly related to hypertension, development of cerebral symptoms, the change in the intensity of carotid bruit, and the degree of original stenosis. Jesse E. Thompson and associates[28] similarly recommend routine study of patients with carotid bruit and advocate carotid endarterectomy in asymptomatic patients with high-grade internal carotid stenosis. Indication for surgery in asymptomatic patients should of course take into consideration the risk of the operative procedure in the respective institution, the cardiorespiratory reserve, and the age of the patient.

The coexistence of a severe asymptomatic internal carotid artery stenosis and symptomatic abdominal aortic aneurysm, coronary artery disease or urgently needed aorto-iliac reconstruction may justify simultaneous carotid endarterectomy and other vascular procedures. We have employed this approach in a number of patients without mortality.

INTRALUMINAL SHUNT

In spite of much debate and careful experimental studies we have found no good substitute for a carefully applied intraluminal shunt. A recent report[11] on the monitoring of stump pressure to predict the need for intraluminal shunting is well worth reviewing. It has been our policy, however, to use intraluminal shunting routinely after systemic heparinization and we have found no reason to abandon this technique. The internal shunt is maintained in place by appropriate clamps.[15] It is important to complete the endarterectomy without haste, making certain that the distal end of the atheromatous plaque is totally removed. We have become convinced that unless the entire distal end of the atheroma is removed, recurrence is quite likely. We have re-explored seven patients with recurrent stenotic lesions of the internal carotid artery, and in every case the site of recurrence was at the terminal end of the endarterectomized segment, suggesting that a piece of atheroma had been left in place in the previous procedure.

EMERGENCY CAROTID THROMBOENDARTERECTOMY

Emergency operation for carotid bifurcation disease is still occasionally indicated; as the experience in this field has increased, the number of patients subjected to emergency surgery has markedly decreased.

Disappearance of bruit following angiography in a patient with severe stenotic lesion requires emergency surgery regardless of the presence or absence of new neurologic symptoms or signs.

Sudden onset of a neurologic deficit during the postoperative phase of carotid endarterectomy in a patient who initially recovers from anesthesia without neurologic abnormalities demands immediate re-exploration of the wound under local anesthesia to investigate the possibility of internal carotid thrombosis. Arteriography is time-consuming in these patients and is contraindicated.

In a patient with repeated frequent transient ischemic attacks, whose internal carotid artery is severely narrowed, urgent carotid endarterectomy is indicated. If for any reason surgery has to be delayed, sodium heparin should be administered intravenously until 3 hours prior to the time of surgery.

ULCERATIVE CAROTID BIFURCATION LESION

We have been aware of the significance of embolic lesions from the carotid bifurcation to terminal branches of the carotid system since the early 1960's and in fact we reported our experience with the carotid ulcers in 1963.[16] We have continued to be aware of the high incidence of ulcerated plaques and the frequency of embolic manifestations, but as yet we have not been able to predict its presence with the accuracy required to justify a recommendation for surgical management in patients who have nonstenotic plaque of the carotid bifurcation. Where the lesion is obvious angiographically and the patient exhibits a history of cerebral infarct on the appropriate side, the indication for surgery is quite clear.

Kollarits and associates[17] recently reported on 70 patients with retinal strokes; 53 of them had a clinical picture suggestive of retinal artery occlusion due to emboli from atheromatous disease of the carotid bifurcation, and 45 of these patients were studied by angiography. Carotid bifurcation atheromas were demonstrated as the probable source of emboli in 43 out of the 45 patients (96 per cent). The striking incidence of carotid lesions in these patients suggested to the authors that most retinal strokes are caused by embolization from carotid bifurcation atheroma. None of these patients had cerebral symptoms, and the indication for surgery was prevention of further retinal embolization.

The presence of ulceration and clot formation on the surface of a carotid bifurcation atheroma cannot be predicted routinely on the basis of angiographic evaluation. We have found ulceration in patients where we did not suspect its presence. We therefore recommend gentle dissection of the carotid bifurcation and careful observation of the lumen prior to the insertion of the intraluminal shunt in order to prevent embolic manifestations. At least 20 per cent of the lesions that are treated surgically have an ulcer with a thrombus lodged in a crater within the atheroma. Dislodgment of cholesterol crystal emboli into the retinal vessels has been reported by Hollenhorst[12] and others.[9, 25, 29] In these individuals neither inspection of the atheroma nor radiologic observation would permit accurate prediction as to the source of platelet, fat or cholesterol

emboli. The mere knowledge of the possibility of an ulcerated lesion would emphasize the tremendous care that is required to avoid puncturing the carotid bifurcation for angiography in order to avoid the dislodgment of atheromatous debris or thrombus.

COMMON CAROTID OCCLUSION

Occlusion of the common carotid artery occurs uncommonly: there was an incidence of 5 per cent in a series of 1400 patients followed in our institution. Left carotid involvement was more frequent than the right at a ratio of approximately 2 to 1. Patients with common carotid occlusion have significant symptoms of cerebrovascular insufficiency and they are in need of surgical management. Total occlusion of the common carotid artery may occur secondary to advanced atherosclerotic involvement of the carotid bifurcation. In these patients the internal carotid artery is often totally occluded and the origin of the external carotid artery narrows down gradually (Fig. 1). Often at completion of this occlusion the stagnant blood in the common carotid artery thromboses and becomes an organized whitish core. The other two causes of carotid occlusion are progression of atheromatous involvement at the origin of the common carotid artery (Fig. 2) or in the mid portion of the vessel (Fig. 3). Patency of

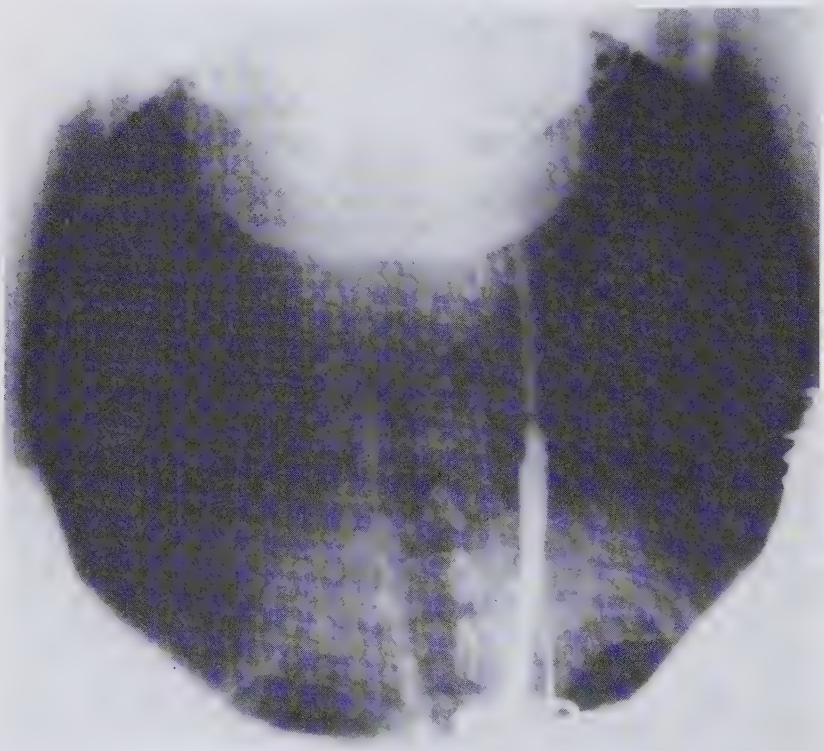

Figure 1. Four weeks after the arteriogram the carotid pulse disappeared. Transient left eye blindness necessitated left carotid thromboendarterectomy.

Figure 2. Severe stenosis of the origin of left common carotid artery.

Figure 3. Marked atherosclerotic involvement of the midportion of the right common carotid artery.

both terminal branches may be preserved in these two instances, making surgical attempts most rewarding.

The standard techniques used to restore circulation involve simultaneous thoracic and cervical approaches for an aorto-carotid bypass graft or, when possible, a carotid-subclavian bypass. In selected patients, however, in order to avoid the added risk of thoracotomy or in the presence of concomitant subclavian artery occlusion, a simpler and more direct approach may be employed. Wesley S. Moore and associates[21] reported their experiences with thrombectomy of the common carotid artery, and they were able to restore flow in 7 out of 10 patients. We have attempted retrograde endarterectomy of the common carotid artery in seven elderly patients without mortality or morbidity and with a 100 per cent restoration of flow. Two endarterectomies were accomplished on the right side, while on five patients the left common carotid artery was endarterectomized from the level of the bifurcation down to the level of the arch.

In each instance the carotid bifurcation is exposed through the standard longitudinal neck incision. The carotid bifurcation and the internal and external carotid arteries are dissected free, and the common carotid artery is freed and mobilized for a distance of 4 to 5 cm proximal to the bifurcation. The atheromatous core is then exposed through a generous longitudinal incision over the carotid bifurcation (Fig. 4). The cordlike

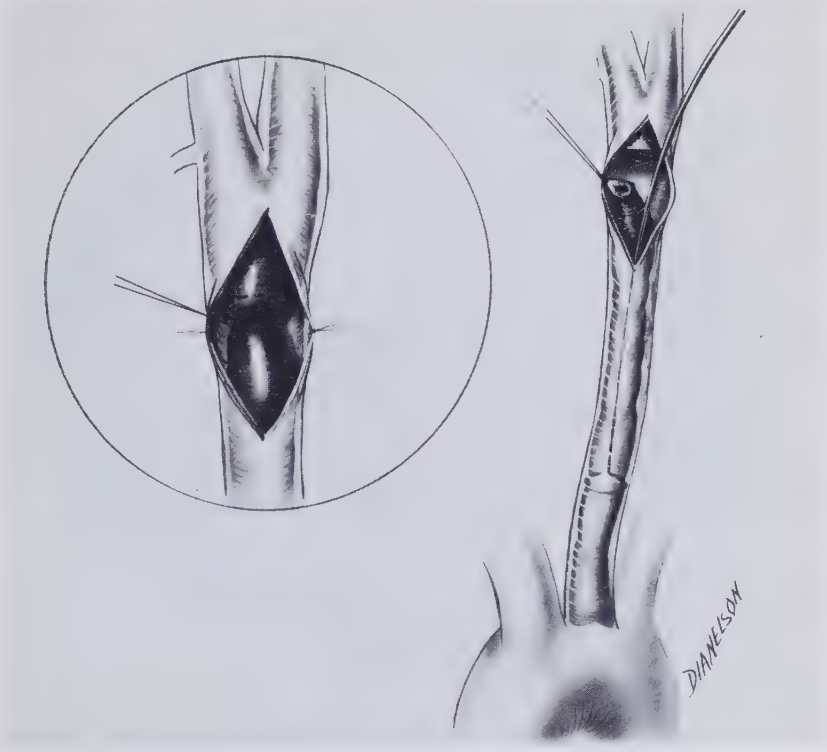

Figure 4. Retrograde common carotid endarterectomy.

structure is encircled by developing the cleavage plane between the atheroma and the arterial wall. The atheromatous cord is transected and the proximal end is carefully freed from the arterial wall. An endarterectomy loop of the appropriate size is then passed over the thrombotic cord. With gentle tension on the cord and downward pressure on the stripper, one can remove the entire atheromatous and thrombotic cord down to the level of the arch. The force of arterial pressure delivers the specimen into the operative field, and one must be prepared to clamp the common carotid artery to control brisk bleeding. The patient is heparinized systemically at this stage to prevent clot formation in the endarterectomized segment. In case of doubt as to the completeness of endarterectomy, passage of a Fogarty catheter will remove residual debris. Distal endarterectomy is then accomplished in the standard manner.

Chronic occlusion of the internal carotid artery is often inoperable, but restoration of flow to the external carotid artery is routinely possible. Where the origin of the external carotid artery is markedly narrowed, widening of the lumen can be accomplished by the use of a flap constructed by division of the internal carotid artery 2 to 3 cm beyond the bifurcation, as shown in Figure 5. Only two patients in this group of seven had recent occlusion. The remaining five had chronic occlusion of

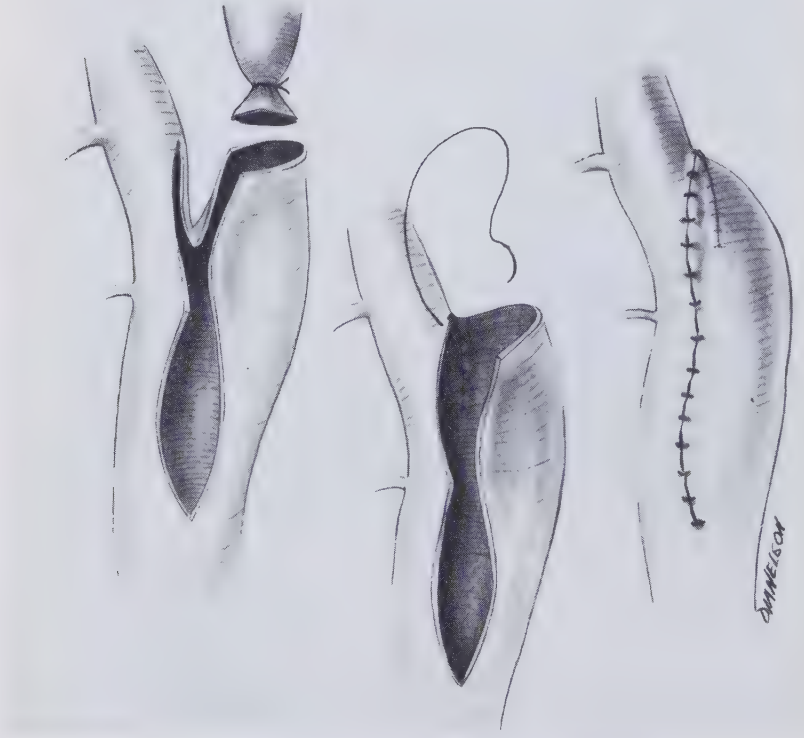

Figure 5. Carotid bifurcation angioplasty.

at least 6 months' duration. All of these patients have been followed; carotid pulses have remained present in each and they are presently all asymptomatic. Because of this recent experience we believe that in patients with significant symptoms of cerebral arterial insufficiency and in the absence of a carotid pulse, the recommendation of carotid bifurcation exploration with the hope of restoration of flow through the external carotid artery or possibly both branches by endarterectomy is worth while. However, extreme care is necessary to prevent tearing of the adventia of the common carotid artery at its origin. If there is any difficulty with the passage of the endarterectomy loop or wire, the procedure should be abandoned and replaced by a subclavian-carotid bypass if feasible.

A broader application of the retrograde common carotid endarterectomy may encompass all cases of partial common carotid occlusion requiring carotid bifurcation endarterectomy. Long-term results of such cases undergoing carotid endarterectomy alone are not satisfactory. Progression of atheromatous disease in the common carotid artery often results in the occlusion of the entire system.

CASE 1. A 60-year-old woman had a left carotid endarterectomy in April 1970 for severe transient ischemic attacks. Narrowing of the common carotid artery was noted on angiogram prior to endarterectomy, but the flow was considered satisfactory. She returned in August 1972 with recurrence of symptoms, and the left carotid pulse was found to be markedly diminished. Angiogram (Fig. 6) revealed

Figure 6. C. R.: Angiogram reveals marked stenosis of the common carotid artery—complete occlusion of the external carotid artery and patency of endarterectomized carotid bifurcation.

marked stenosis of the common carotid artery with satisfactory flow through the endarterectomized carotid bifurcation. Following angiography she lost her carotid pulse and as an emergency procedure retrograde common carotid endarterectomy restored flow to internal carotid artery.

We believe that in patients with advanced atheromatous involvement of the common carotid artery where the lesion extends proximally beyond reach, removal of the atheromatous core by retrograde endarterectomy will provide better flow and may prevent recurrent occlusion by progression of disease.

SUBCLAVIAN ARTERY OCCLUSION

The proximal end of the subclavian artery is by far the most common site of occlusion, sparing the origin of the vertebral artery and the cervical branches. Reivich and co-workers in 1961 described a phenomenon unappreciated before which involved the subclavian artery. Significant obstruction of the proximal end of this vessel not only decreases cerebral blood flow by diminishing forward flow, but also causes reversal of basilar-vertebral flow when the pressure in the subclavian artery distal to the occlusion drops below the pressure in the basilar artery (subclavian steal syndrome). When the innominate artery is occluded the right carotid as well as the subclavian flow become reversed.

The incidence of subclavian artery occlusion in our series is 10 per cent, with the left subclavian artery being more frequently involved at a ratio of approximately 2 to 1. One hundred thirty-nine patients exhibited clinical findings of subclavian artery occlusion. Twenty-three patients had bilateral disease. Fifty-four patients had complete occlusion of the vessel, and six of these patients had total occlusion of both subclavian arteries. Forty-five of these patients had involvement of the carotid system as well. Seventy-two patients in the series had surgical management to restore subclavian arterial flow.

Indications for surgery were significant cerebral or upper extremity symptoms, or both. In patients with concomitant carotid bifurcation disease, carotid endarterectomy was accomplished first. The majority of these patients became asymptomatic and did not require operation for the subclavian occlusion. It was uncommon for the upper extremity and cerebral symptoms to be present concurrently. The classic subclavian steal syndrome was noted in only 15 patients, in whom arm exercise caused cerebral symptoms. In six individuals the subclavian disease extended distally to the level of the axilla. These patients had disabling arm symptoms. Restoration of flow was accomplished by carotid axillary bypass in one individual and aorto-axillary bypass in the other five patients.

Carotid subclavian bypass is the preferred method of restoration of flow and was used in 47 patients in our series. Twelve of these patients had simultaneous carotid endarterectomy and a carotid-subclavian bypass.[23] There was no hospital mortality in this group. There were four late thromboses of the graft, two wound infections, and two false an-

eurysms. One patient developed stroke following carotid endarterectomy and bypass. Wound infection is the most serious complication in the presence of a synthetic graft. One of these two patients responded well to antibiotics and adequate drainage of the wound and it was possible to preserve the functioning graft. In the other case it was necessary to remove the Dacron graft. Aorto-subclavian bypass was accomplished in 18 patients with one death due to myocardial infarction and six late thromboses. Restoration of flow by carotid-subclavian bypass was accomplished in four of these patients.

Patients with complete occlusion of the common carotid artery and subclavian artery on the same side present challenging problems. These patients are usually severely symptomatic and require surgical management. Subclavian-subclavian bypass[8, 10] and subcutaneous axillary-axillary graft[22] reported by three independent groups may be employed to advantage in these patients.

Case 2. A 45-year-old man was studied about a year ago for transient bilateral blurring of vision, episodes of left eye blindness, slurring of speech, numbness of the right side of the face and arm, and marked dizziness. Selective brachiocephalic angiography revealed complete occlusion of the left subclavian artery and left common carotid artery, severe stenosis of the right internal carotid artery, and vertebral subclavian steal. Carotid bifurcation endarterectomy was performed on the right side initially but did not relieve cerebral symptoms. Visual difficulty and dizziness persisted.

Retrograde left common carotid endarterectomy as described before was accomplished at a later date. The internal carotid artery on this side was totally occluded. The external carotid branch was small but contained pulsatile flow through collaterals. The endarterectomized common carotid artery was transected at the level of bifurcation and the proximal end was passed behind the sternocleidomastoid muscle and was anastamosed to the side of the subclavian artery through a supraclavicular incision (Fig. 7). An arch angiogram 8 months later reveals a patent right internal carotid artery and the satisfactory flow through the endarterectomized left common carotid artery supplying forward flow into the left vertebral and subclavian arteries (Fig. 8).

VERTEBRAL ARTERY LESION

Although the incidence of vertebral artery occlusion is significant, direct surgery on this vessel is rarely required. Patency of one vertebral artery is sufficient for adequate basilar flow, and where vertebral lesions are coexistent with carotid bifurcation disease, restitution of carotid flow often relieves the symptoms of vertebral basilary insufficiency. However, it is occasionally necessary to alleviate the stenotic lesion of the proximal end of the vertebral artery, and this is best accomplished in our opinion with vein patch angioplasty. We have employed this procedure without the use of a shunt or other modalities for protection of the brain on 20 patients with restoration of flow in 19 patients and without an incidence of stroke or death. Aggravation of preoperative neurologic deficit occurred in two patients, with satisfactory recovery within 2 weeks. Both patients had internal carotid occlusion and stroke several months prior to vertebral angioplasty.

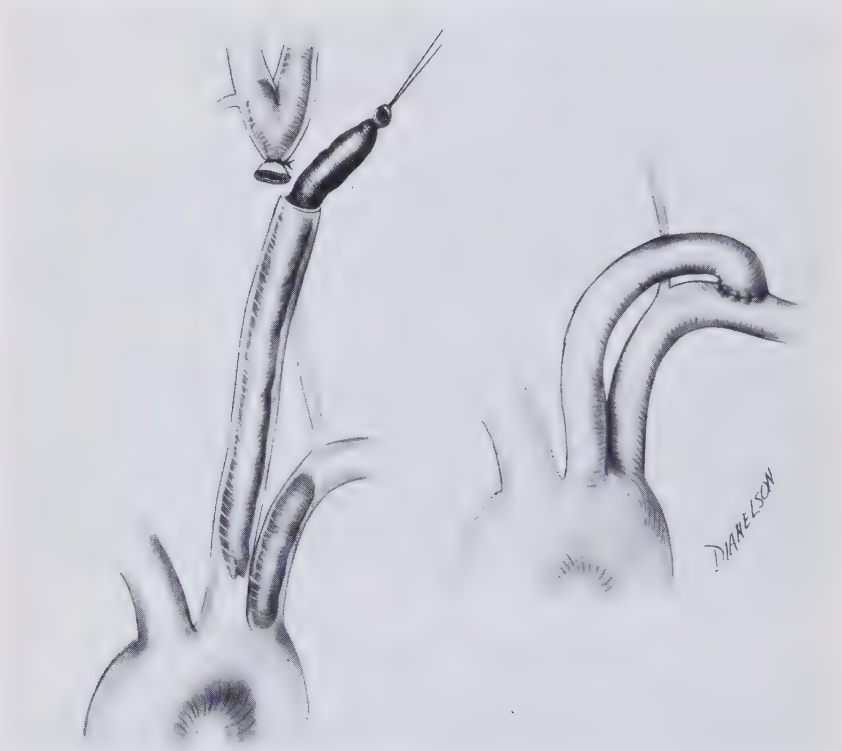

Figure 7. Aortosubclavian bypass employing endarterectomized left common carotid artery.

Figure 8. Arch angiogram reveals complete left subclavian occlusion and aortosubclavian bypass employing endarterectomized left common carotid artery.

CONCLUSION

Atherosclerotic lesions of the extracranial portion of the brachiocephalic system are segmental in nature and amenable to surgical procedures currently available. The value of carotid endarterectomy in palliation of symptoms and in prevention of stroke has been demonstrated. Reconstructive procedures for subclavian, common carotid, and innominate artery occlusion were attempted only in the presence of significant cerebral or upper extremity symptoms.

Mortality and morbidity of these operations have been reduced to a very satisfactory level: an overall mortality of 2 per cent and a stroke incidence of 1.8 per cent. Eighty-five per cent of patients treated have benefited significantly from surgical management.

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Changing Concepts in Renovascular Hypertension

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The history of renovascular hypertension (RVH) can be divided into four eras. The first era was initiated by Goldblatt's experimental demonstration that renal artery constriction resulted in atrophy of the kidney and concurrent development of hypertension.[15] In that era, patients with hypertension and a small kidney, as determined by intravenous pyelography, were treated by nephrectomy. Arteriograms were seldom done. In 1956 Smith reviewed 575 such cases treated by nephrectomy and found that only 26 per cent were benefited.[26] The conclusion was that nephrectomy should be done only for strict urologic indications with no thought of influencing the blood pressure.

The second era overlapped the first. In the early 1950's vascular surgery started to develop and aortograms were performed with increasing frequency. Renal artery stenosis was demonstrated in many patients and most of them had high blood pressure. In this second era, patients with hypertension and renal artery stenosis were treated by arterial reconstruction or nephrectomy. However by 1960 it was apparent that less than 50 per cent of patients so treated were benefited. Many physicians and surgeons became disillusioned with the concept of renovascular hypertension. In many centers this disillusionment persists today.

The third era similarly overlapped the second. In 1957 Howard and his associates described a test which would indicate when renal artery stenosis was the cause of the patient's hypertension.[3] It was based on differential comparison of the urine from the two kidneys and an increased resorption of water and sodium by the involved kidney. The test was later modified to include an increased concentration of creatinine in the urine of the involved kidney.[17] Stamey devised a similar test which included os-

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motic diuresis and an infusion of para-aminohippuric acid which also appeared in increased concentration in the urine from the involved kidney.[27] Rappaport and Birchall also devised differential urinary function studies.[2, 24] For a number of reasons these tests never achieved widespread acceptance. They require cystoscopy and ureteral catherization, and patient discomfort and morbidity are not inconsequential. Many urologists are not anxious to do the tests. Their casual performance yields confusing and inaccurate results. If split renal function studies (SRFS) are done with meticulous care they yield highly accurate results, and post-examination morbidity is largely limited to suprapubic pain, flank pain and colic in the first 24 hours following the tests.[12, 22] The third era also witnessed the development of reliable renin assays.[21] Peripheral plasma renin determinations did not prove to be particularly helpful in the recognition of RVH, but comparative assessment of renal venous renin activity (RVRA) proved to be highly accurate in the recognition of RVH.[10]

Significant experience with the use of these tests (SRFS and RVRA) in the selection of patients for operative treatment of RVH has now accumulated.[6, 10, 11, 22] Many patients have been carefully followed for 5 to 10 years following successful renal arterial revascularization. The follow-up has included serial renal arteriography in a sizable number of patients. It is now possible to retrospectively analyze the data from these patients and come to some conclusions, at least tentatively, regarding the recognition and treatment of RVH, and this is the fourth era in the history of RVH. This era, currently in progress, also involves improvement in operative techniques, identification of hypertensive patients who are candidates for intensive evaluation, comparative evaluation of operative versus drug treatment of RVH, and finally the difficult problem of the patient with bilateral renal artery stenosis.

The following observations are based on a study of 1070 consecutive hypertensive patients and on experience with 122 patients who have had operative treatment for RVH and a minimum follow-up of 18 months. Most of the basic data has been reported elsewhere, and only selected items, related to the current topic, will be included.[10]

CLINICAL CHARACTERISTICS

The history and physical examination provide little helpful information in the recognition of the patient with RVH or in differentiating the patient with "essential hypertension" (EH). The patient with RVH is usually of normal or less than the normal body weight; rarely is he obese.[16] A family history of hypertension or a history of recent acceleration and duration of hypertension have not been helpful in differentiating RVH and EH. Even the age of the patient does not have predictive value except in the first decade of life. Children under 10 years of age who have hypertension are very likely to have RVH, a 90 per cent chance in our experience. The next highest incidence was in patients 60 to 69 years of age, where 33 per cent have had RVH.[7]

Several recent reports have suggested that RVH in patients over 50 years of age should be treated with antihypertensive drugs because the results of operative treatment in this age group are poor and the morbidity and mortality excessively high.[5, 19, 25] Forty-three per cent or 52 of the patients in our group of 122 were over 50 years of age and over 80 per cent of them were either cured or improved following operative treatment.[10] There were two operative deaths in these older patients, who had operative treatment limited to renal arterial revascularization. If one adds simultaneous operative treatment of aortic aneurysmal or occlusive disease, a higher operative mortality is encountered.

The only feature in the physical exam that has been helpful is the frequency of abdominal bruits (50 per cent) in the patients with RVH. Examination of the eye grounds and heart can give some estimate of the severity of the hypertension but no information regarding the underlying etiology. In a similar manner the routine urinalysis, hemogram and serum electrolyte determinations are not particularly helpful. Urinalysis and urine cultures are necessary to identify patients with underlying renal parenchymal disease. Serum potassium levels are often low in patients with severe RVH, and may reflect secondary aldosteronism. But many, if not most, patients have been on diuretic therapy, a common cause of hypokalemia in the hypertensive patient.

SPECIAL DIAGNOSTIC PROCEDURES

A number of special studies have been advocated for the recognition of the patients with RVH. They include the rapid sequence excretory urogram, renography, kidney biopsy, measurement of pressure gradients across the renal artery stenosis, renal arteriography, and the already mentioned SRFS and RVRA. The reader is referred to other publications for information on the limitations of renal biopsies and pressure gradients.[19, 29] As regards renography, thus far it has proved to be of limited help. At present we are evaluating a triple isotope renogram but have not been encouraged with the results. This presentation will limit itself to discussion of the IVP, arteriogram, SRFS and RVRA.

Rapid Sequence Intravenous Pyelogram

This test has been recommended as a screening study for RVH. Criteria for the interpretation of the test have been detailed elsewhere.[7] In some centers if this test is normal further studies are not done. In our experience the IVP has given a false negative result in 31 per cent of the patients with RVH.[10] The fact that half of these false negative results were in patients with bilateral lesions explains in part the high incidence of false negative results, but does not mitigate against the fact that if one is relying on this screening test to detect RVH many cases will be missed.

Renal Arteriography

Our experience has led to the policy that renal arteriograms should be done in any hypertensive patient in whom operative treatment would be

recommended if a significant lesion were found. This eliminates the mildly hypertensive patient and also the unacceptable operative risk. As a general rule we have required that the patient have documented diastolic blood pressures in excess of 100 mm Hg, and the majority have had pressure in excess of 120 mm Hg. Estimation of operative risk in a patient with extrarenal vascular disease is much more difficult. If the hypertension is posing the greatest threat to the patient's life (for example, aggravating coronary heart disease, congestive failure, cerebrovascular problems or unrelenting damage to the contralateral kidney), then intensive evaluation including arteriography is recommended.

There have been several important changes or developments relating to renal arteriography in the past 5 to 8 years. A simple abdominal aortogram in the anteroposterior projection is not sufficient. The renal artery often originates on the anterior or posterior wall of the aorta, and lesions involving the orifice of the renal artery can be hidden in the AP projection (Fig. 1). The distal renal artery courses in an almost AP plane, and lesions situated in this segment can be masked in an AP aortogram (Fig. 2). Selective and oblique renal arteriograms are required to accurately delineate the true morphology. An important piece of evidence supporting this view is found in the patients with fibromuscular dysplasia of the renal arteries. We reported in 1969 that these lesions were unilateral in 60 per cent of the cases and bilateral in the other 40 per cent.[8] Today, with selective and oblique arteriograms the incidence of bilateral lesions is found to be over 90 per cent.[10]

Other refinements in the performance of renal arteriography include subtraction and magnification arteriography. We have experience with the former and find it to be helpful; we have little experience with the latter.

Renal arteriography carries some risk, and concern about this risk still deters many physicians in the study of hypertensive patients. We have done renal arteriographic studies in over 1400 hypertensive patients with no deaths and 10 significant complications, each of which was correctable.[12] These were patients with no significant extrarenal vascular

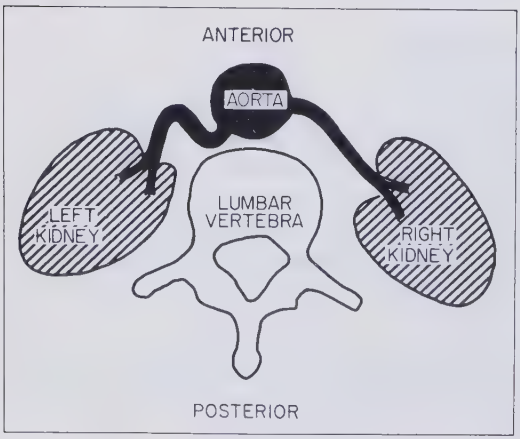

Figure 1. Schematic drawing showing the origins and the course of the renal arteries. Arteriograms made in the straight anteroposterior projection may fail to demonstrate lesions at the orifice of the arteries or in the distal arteries.

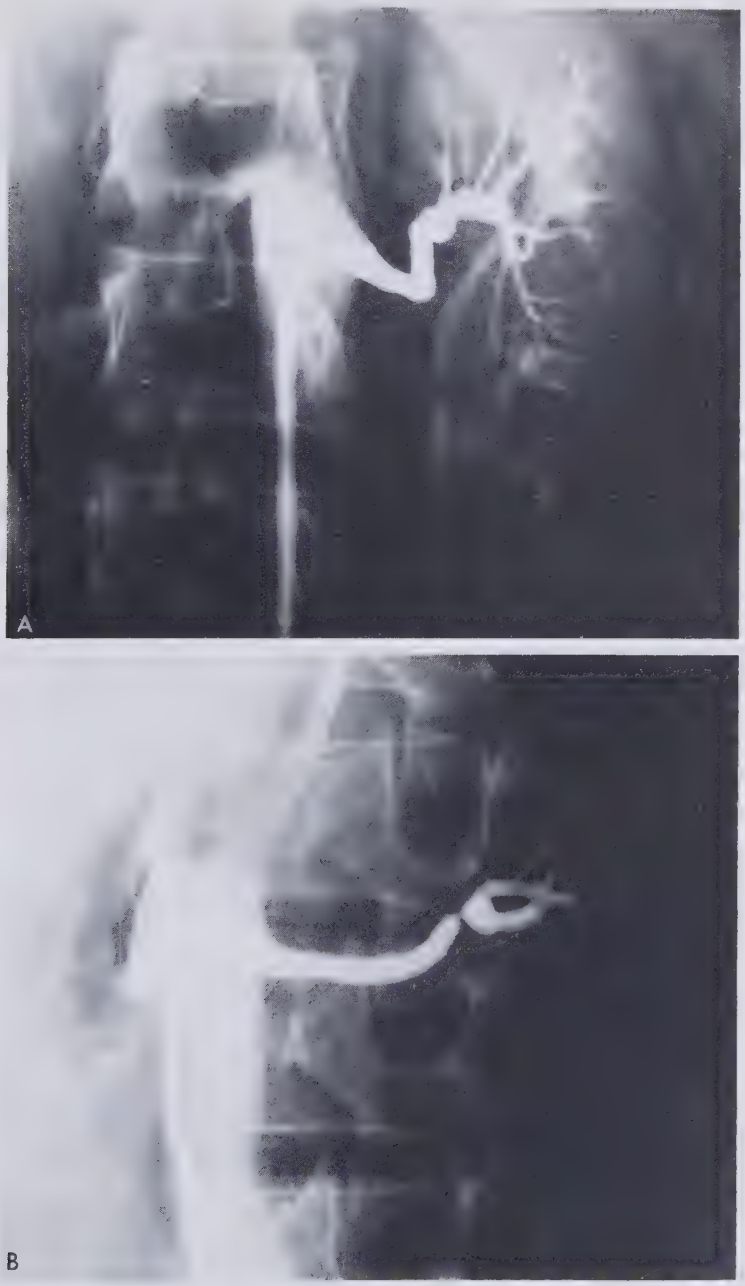

Figure 2. *A*, Selective arteriograms in anteroposterior projection and *B*, in the oblique projection. Lesion is hidden in *A*.

disease. If patients with aneurysmal or severe occlusive disease of the aorta are included, then the risk is greater, but such patients are at greater risk due to their underlying disease.

Renal Venous Renin Assays and Split Renal Function Studies

In our experience if either of these studies indicates that a renal artery stenosis is functionally significant, a good response following operative treatment can be expected in greater than 90 per cent of the patients.[10] However the criteria used for interpreting the results of the tests have been liberalized. Instead of the classic criteria discribed by Howard and Stamey, we consider consistent decreases in volume and increases in creatinine and PAH concentration from the involved kidney to constitute a positive test.[13] A ratio of RVRA from the involved:uninvolved kidneys of 1.5 or greater has been the usual criterion for a positive test. Our data now indicate that a 1.4 ratio suffices to identify a patient with RVH.[13]

Now that reliable renin assays are widely available many centers have come to rely solely on this test in the diagnosis of RVH and have stopped doing SRFS. This may be ill-advised. We have a group of some 44 patients who had hypertension and unilateral renal artery stenosis and who are unequivocally cured following operative treatment. Eight per cent of them had normal SRFS and 10 per cent had normal RVRA.[13] Obviously these are not the same groups of patients, because one of the tests had to be positive in order for the patient to be selected for operative treatment. Because of these data we continue to do both tests in each patient with renal artery stenosis and recommend operative treatment if either test is positive. O'Connor and Simon have previously written in support of continued use of split renal function studies in the study of patients with suspected RVH.[22]

OPERATIVE TECHNIQUE

Several aspects of operative technique remain somewhat controversial, or at least unsettled. They include choice of graft material, end-to-side versus end-to-end anastomosis between graft and renal artery, continuous versus interrupted suture techniques, and endarterectomy versus bypass graft in atherosclerotic lesions. Wylie and his associates prefer autologous hypogastric artery whereas we have chosen to use autologous saphenous vein for aortorenal grafts, as have Fry and DeCamp and their associates.[4, 6, 30] However, long-term serial arteriograms in patients with saphenous vein grafts are showing progressive dilatation of the saphenous vein in some patients (Fig. 3). Ernst and Fry first reported this finding.[6, 28] In their latest report there were 74 vein grafts examined 8 to 109 months after implantation. Six of them had develooed aneurysmal dilatation. Of 60 veins that we have followed with periodic arteriograms over 1 to 8 years, two have developed aneurysmal dilatation. Progressive dilation to the point of rupture has not, to our knowledge, been reported. Changes shown in Figure 4 are the most severe that we have encountered thus far. Similar serial follow-up arteriographic stu-

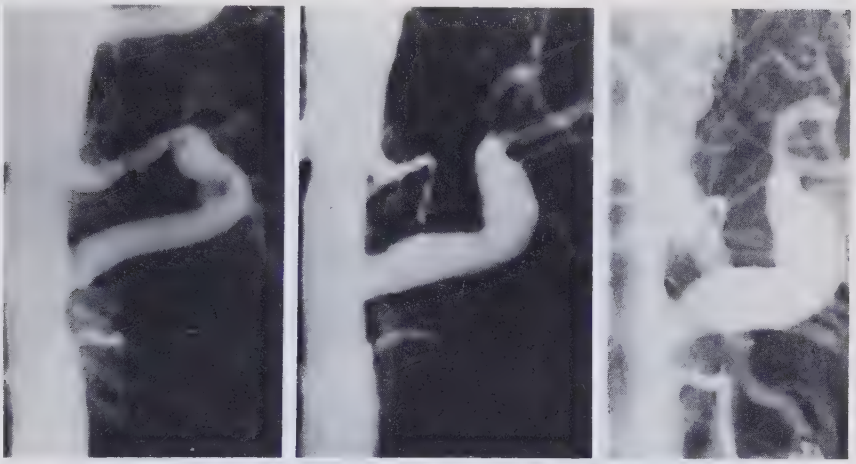

Figure 3. Arteriograms showing a saphenous vein, aortorenal bypass at 1, 3, and 6 years postoperatively. Aneurysmal dilatation of the vein has developed. A recent arteriogram shows no further dilation at the end of the 7th year.

dies of autologous hypogastric arteries used as aortorenal grafts have not been reported but are badly needed. The fact that most renal revascularizations are being done in relatively young patients lends urgency to the need for comparative follow-up studies. It may be that autologous artery will prove to be clearly superior. Dacron aortorenal bypass grafts are very satisfactory in selected cases, in which the distal renal artery has a large diameter, as is often the situation in atherosclerotic renal artery stenosis (Fig. 4). Our experience with endarterectomy of atherosclerotic lesions is too limited to allow comment; others strongly advocate it.[30]

Theoretically, an end-to-end anastomosis between the graft and the renal artery might seem to be hemodynamically preferable to an end-to-side anastomosis. However the end to side technique allows the creation of a larger anastomotic lumen. As a rule, we have employed this latter technique, as has DeCamp, while Wylie and Fry have used an end-to-end anastomosis.[4, 6, 30]

Exposure of the renal arteries is one of the more challenging aspects of the surgery of RVH. At present the left renal artery is approached by exposing the aorta, dissecting along the aorta up to the left renal vein, and then dissecting out the artery above or below the vein. The right renal artery, especially in lesions involving the distal or middle portion of the artery, is exposed by mobilizing the duodenum and right colon medially and then dissecting out the renal pedicle. The course of the bypass graft, whether it be antecaval or retrocaval on the right or in front or behind of the left renal vein, varies from patient to patient. The anatomy and course of the renal artery and the configuration of the renal artery lesions vary from case to case and often dictate the technique to be used. In some cases an end-to-end anastomosis is clearly indicated. A continuous suture technique should be avoided because of the pursestring effect which may narrow the anastomosis. We have chosen to interrupt the anastomosis in

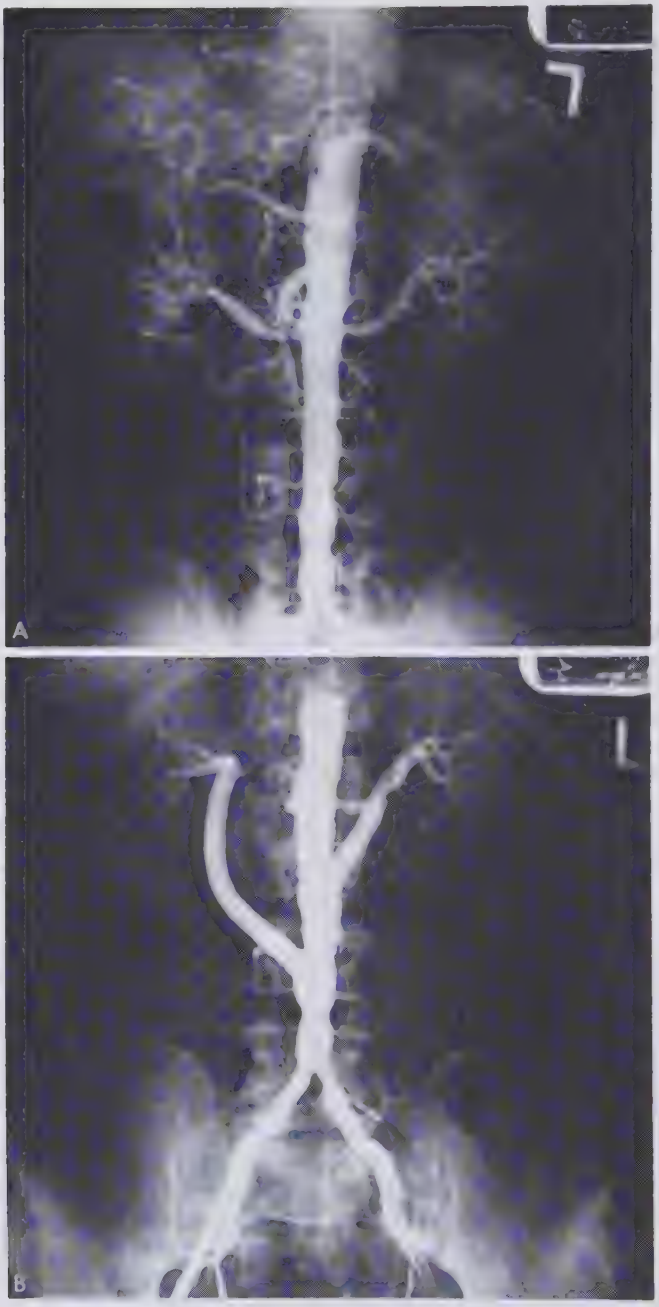

Figure 4. *A*, Aortogram showing bilateral atherosclerotic stenoses of the renal arteries and post stenotic dilatation. *B*, Aortogram 1 year following bilateral Dacron aortorenal grafting.

Figure 4 continued on opposite page.

Figure 4 *Continued.* *C* and *D*, Aortogram 10 years postoperatively. Although the aortic atherosclerosis has progressed, the grafts remain patent.

four places but have not used a completely interrupted suture technique. In a comparative study of a monofilament polypropylene suture (Prolene) and a braided synthetic suture, blood loss from the suture lines was twice as great with the braided material. Microscopic examination of the braided suture reveals a serrated surface while the monofilament suture had a smooth surface. This difference in blood loss was attributed to a sawing effect which occurs when the braided suture is pulled through the arterial wall with an attendant enlargement of the needle hole.[10]

Thrombosis of aortorenal grafts has been reported as occurring in 5 to 35 per cent of cases.[12] Many factors are involved in these graft failures, including those related to operative technique and choice of materials. In addition the nature of the arterial lesions plays a role. In the early 1960's nephrectomy was often done in patients with fibromuscular lesions. However, we now know that fibromuscular dysplasia of the renal arteries is usually bilateral, and frequently the severity of the lesions progresses with the passage of time. Nephrectomy should not be done in these patients except under the most demanding circumstances, such as uncontrollable life-threatening hypertension. Fibromuscular lesions often extend out to and into branch renal arteries. The risk of graft thrombosis is appreciably greater in such patients. Experience shows that thrombosis of an aortorenal graft is largely limited to the early postoperative period.[6, 10] For a number of years we used the postoperative IVP to gain reassurance about the patency of the revascularization. It still has some value, for example if the IVP laterilized preoperatively and does not postoperatively. However we have a number of cases in which the preoperative and postoperative IVP's were indistinguishable and in whom arteriography showed the graft was thrombosed (Fig. 5). The mere findings of a functioning kidney does not assure graft patency. An IVP which shows a nonfunctioning kidney does usually indicate a graft failure. But even this is not completely reliable. Reversible tubular necrosis resulting in nonfunction of the kidney has been documented by IVP's at 1, 2, and 3 weeks postoperatively with return of normal function at the end of the fourth postoperative week.

Renal artery reconstruction usually involves a period of renal ischemia. The effects on the kidney of 10 to 20 minutes of ischmia are not well documented. In the usual case the contralateral kidney is essentially normal so that serum urea nitrogen and creatinine levels are not helpful in recognizing renal injury. We have evidence from renography that even short periods of renal ischemia causes considerable derangements in renal function. Renal transplantation experience has shown that flushing the blood out of the renal vasculature and cooling of the kidney provides considerable protection. At the present time we are attempting to evaluate the merit of these maneuvers in patients undergoing renal artery reconstruction and in experimental animals as well. After the renal artery is occluded, the vasculature is flushed with 500 ml of a cold solution (3 to 4° C) of Ringer's lactate containing heparin. The perfusate is collected from the renal vein to prevent its return to the systemic circulation. It is too early to evaluate the merit of this technique.

Another development is the use of a similar technique to remove the kidney. The technique, as we have used it, involves division of the renal

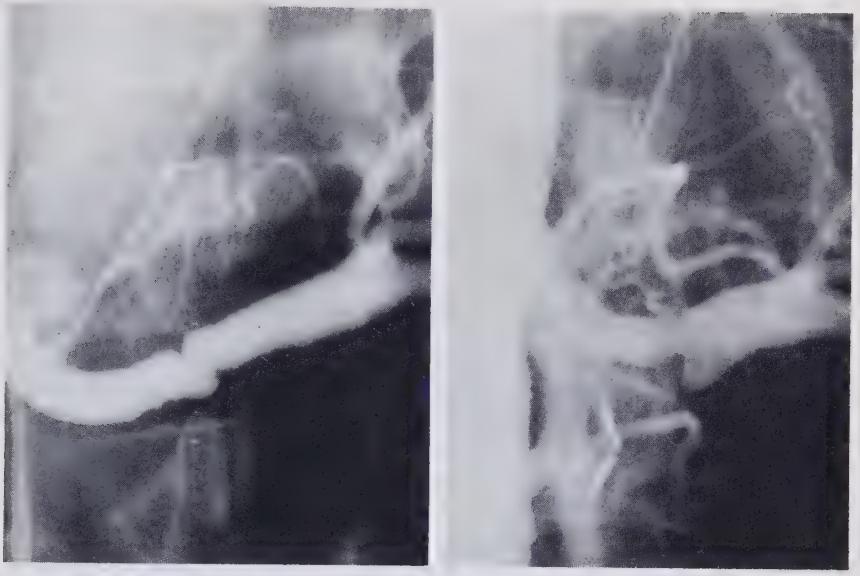

Figure 5. On the left is a preoperative arteriogram showing fibromuscular lesions of the left renal artery. A saphenous vein aortorenal bypass was done. One year later (right), the vein graft had thrombosed but the renal artery remained patent.

artery and vein but not the ureter, delivery of the kidney onto the abdominal wall, flushing and cooling of the kidney, repair of the renal artery lesions using magnification and microsurgical techniques and then autotransplantation of the kidney. We have now done this in six patients, one of which had bilateral ex vivo repair in two stages, with documented anatomical success in each instance. This is not an original technique, several others have reported it previously.[1, 20, 23] Again, it is too early to present an evaluation beyond early success.

Dilation of branch artery stenosis has been recommended by Fry and his associates.[14] This technique has been used in conjunction with bypass operation in a few of our patients. Our experience is too limited to allow evaluation at this time. On the other hand we believe that one does not have to bypass every fibromuscular lesion in a renal artery in order to obtain a salutory blood pressure response. On a number of occasions the renal artery anastomosis has been placed into a diseased segment of the artery or placed proximal to an isolated branch artery lesion with a good result.

Although there are still many unanswered questions regarding renovascular hypertension, bilateral renal artery stenosis is among the most troublesome. In 70 per cent of the cases one side has been dominant and the SRFS and RVRA have lateralized to that side.[10, 11] This still leaves 30 per cent of cases with bilateral lesions in which one does not know whether one, both or neither of the lesions cause the patient's high blood pressure. In such cases the following policy has been adopted. If the diastolic hypertension is mild to moderate (< 120 mm Hg) and easily controlled with drugs, this has been the mode of treatment. If the hyperten-

sion is severe (> 120 mm Hg) or difficult to control, then the side which appears to be more severely involved on the arteriogram is repaired and the SRFS and RVRA are repeated in the postoperative period. If hypertension persists and the studies lateralize to the contralateral kidney, the artery to that kidney is repaired. It can be argued that one should proceed with repair of both arteries at the same time and avoid a second operation. This might appear to be the simple solution, but the question is considerably more complex. The discussion of this question is usually clouded with statements regarding preservation of renal function irrespective of blood pressure response. This view is supported by the fact that renal arery stenosis, regardless of etiology, can progress to complete occlusion of the artery and in some instances destruction of the kidney. However there is little reported objective evidence relating to the frequency or rapidity of such progression. Also pertinent to this discussion is the frequency of thrombosis of a reconstructed artery. Our experience leads us to believe that the incidence of reconstruction thrombosis may approximate the incidence of significant progression of a mild to moderate renal artery stenosis. However, this has not been established. There are some patients with severe bilateral renal artery stenosis who will benefit from bilateral reconstruction. In most instances, in such a case, a two-stage approach as outlined above is preferred to a simultaneous bilateral reconstruction. Just as indiscriminate repair of all renal artery lesions in patients with hypertension, as was done in the second era of the history of RVH, led to disappointing results and to dissillusionment with the whole concept of RVH on the part of many, a similar approach to patients with bilateral stenosis is likely to have similar consequences.

The final area of controversy concerns[3] the patient with RVH who is 50 years of age or older. Many physicians feel that such a patient should be treated with antihypertensive drugs.[5, 19, 25] Our experience, as stated earlier, is to the contrary.[10] However a prospective randomized study comparing operative treatement with drug therapy will be required to answer this question. At present we are attempting to do such a study. Patients over 40 years of age with unilateral renal artery stenosis, hypertension and positive SRFS/or RVRA are prospectively randomized into drug therapy or operative treatment. It will require several years to obtain an answer to this preplexing question.

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